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OF THE

PHILADELPHIA NEUROLOGICAL SOCIETY.

VOLUME II.

1892—1893.

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PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, October 24, 1892.

The President, Dr. FRANCIS X. DERCUM, in the chair.

Dr. WM. H. MORRISON read a paper entitled

REPORT OF A CASE OF LARGE INTRA-CRANIAL TUMOR (WEIGHING FIVE OUNCES) COMPRESSING THE LEFT FRONTAL LOBE.

A. C., colored, about forty years of age, came under the author's observation in April, 1886. At this time he was a coachman by occupation, but had been in the army and had also served as a sailor, and was said to have suffered shipwreck on one or two occasions. He complained of pains in the head, which were attributed to localized syphilitic meningitis, and he was placed upon iodide of potassium. He soon passed from the author's observation, and was not seen again until two days before death. In the summer of 1888 he had an epileptiform convulsion, which was the only convulsion in the history of the case. The pains in the head returned and increased gradually in severity, and were especially marked on stooping. In August, 1889, he gave up his work, but for a month before this his friends had noticed that his speech was slower than usual. Shortly after quitting work his eyesight began to fail, and soon he was unable to recognize persons or objects at a short distance from him. The pain seemed to be more in the occipital region, although on stooping there would be pain felt in the frontal region. During the last six or nine months of the history the pain was not so severe, and at no time does it seem to have been a marked symptom. A few months after quitting work he was admitted to the Pennsylvania Hospital, where he remained six weeks.

An examination of the eyes showed no light-perception. In the right eye there is extensive hemorrhagic

retinitis, with superficial and deep hemorrhages scattered over the fundus, rendering it difficult to see the disc. The margin of the disc is obliterated. There is extensive œdema. Near the macula are several yellowish white patches, suggestive of hemorrhage. The condition of the left eye is about the same.

After leaving the hospital he received no medical attention. The principal trouble observed by the patient and his friends was the loss of eyesight, which had sunk to simple perception of light. During the day he sat about the house, and at night he was able, unaided, to go up three flights of stairs. In fact, a few days before his death, he dressed himself and came downstairs without assistance. There was no motor disturbance in any part of the body—no weakness of the muscles. Intelligence did not seem to be materially impaired. His memory for past events was retained. In illustration of this it may be stated that two weeks before his death he was visited by friends whom he had not seen for years, and conversed with them intelligently and rationally about events that had occurred ten or more years previously. For recents events, however, memory was defective. The drawling speech continued, and to this was added slight amnesic aphasia. Occasionally he would be unable to remember a certain common word which he wished to use, but when it was suggested he would recognize it and be able to pronounce it without difficulty. Hearing was unaffected; the sense of smell was good, but whether or not there was any difference on the two sides could not be ascertained; the bowels and bladder acted normally.

The writer was called to see him at midnight on December 24, 1891, the statement being that he was in a stupor from which he could not be roused. When seen, however, he was awake. He protruded the tongue normally. He lifted first one arm and then the other, and moved the legs at command. He had high fever, injected conjunctivæ, and other symptoms of influenza, which was prevalent at that time. The following day his condition was about the same; but next day he passed into a comatose condition, and died that evening.

The autopsy was made fourteen hours after death, the examination being confined to the head. The body was that of a stout, well-nourished man, with nothing abnormal on inspection. The scalp was fully one-half inch in thickness, there being a quarter-inch layer of fat; the bone was of normal thickness, and the dura but slightly

adherent to its under surface; the dura was found strongly adherent to the pia mater along each side of the longitudinal fissure, and a portion of it was removed with the brain. A large tumor was at once evident in the left frontal region. The brain and tumor were removed as carefully as possible. It was found that the tumor had eroded the left half of the cribriform plate of the ethmoid, so that there was a direct communication between the interior of the skull and the nasal cavity.

From the inner surface of the frontal bone, one inch to the left of the median line and one inch above the orbital plate, there sprang a conical projection of bone; this had a base one-half inch in diameter, and projected three-eighths of an inch above the surface of the bone. Corresponding to this exostosis, there was a depression in the dura mater and in the new growth. Inspection of the external surface of the bone showed no depression and no evidence of injury.

Examination of the brain and tumor, after removal, showed that the growth had occupied the greater part of the left anterior fossa of the skull. The mass is irregularly pyramidal in shape, its base, corresponding to the left half of the frontal bone, being attached to the inner surface of the dura. The base of the growth measures two and one-half inches vertically by three and one-quarter inches transversely. The tumor extends backward into the frontal lobes two and three-quarter inches. After preservation in alcohol for five months, the weight of the mass is five ounces, its volume nine cubic inches, and it displaces five fluid ounces of water. The orbital plate of the frontal bone was not displaced, and there was no exophthalmus. The tumor was not adherent to the brain-tissue, but seems to be a dural tumor originating at the position of the projection of bone already mentioned, and thence extending gradually, pressing backward the frontal convolutions. Internally and externally the tumor was covered by cerebral tissue; but underneath, the brain-tissue has been displaced and absorbed in the position of the cribriform plate, and the bone itself has been destroyed. The tumor was readily lifted from its cavity, and, in fact, care was necessary to prevent it from falling out when the brain was removed.

The growth was examined microscopically by Dr. C. W. Burr, who pronounced it to be a sarcoma.

The special features of this case, as elicited by post-mortem study, are: The evidences of a slowly-developing

and steadily-progressing lesion, accompanied by pain in the head, not distinctly localized, not especially severe; a single epileptiform convulsion, neuro-retinitis, loss of memory, slowness of mental processes, slowness of speech, and slight amnesic aphasia. To these may be added absence of motor disturbances, and absence of signs of involvement of the special senses other than the sight.

Referring to lesions in this region, Dr. Seguin says:[1]

"Focal lesions of the frontal lobe produce no specific symptoms, and cannot be directly diagnosticated unless they extend as far caudad as the base of the second or third frontal gyri. The forward mass of the frontal lobe including the orbital lobule appears to be inexcitable and insensitive. Even psychic symptoms do not necessarily appear after the loss of a considerable amount of cerebral substance from this region. The diagnosis of tumors, abscesses, etc., in this part of the brain must be made by taking into consideration the seat of the pain, the presence of cicatrices or other etiological indications, the general signs of cerebral irritation and compression, but, after all, usually by exclusion. In some cases unilateral anosmia is produced."

Dr. Mills, speaking of lesions in this part of the brain, says:[2]

"Lesions of the prefrontal lobe, although this is one of the so-called latent districts of the brain, have, in a large percentage of the carefully-studied cases, shown distinct manifestations. The symptoms are largely psychical, and, unfortunately, the physician is not usually well trained to study such phenomena. Mental disturbances of a peculiar character occur, such as mental slowness and uncertainty, want of attention and control, and impairment of judgment and reason. Closely studied, the inhibitory influence of the brain both upon psychical and physical action is found to be diminished. Memory is not seriously affected, although a continuous train of thought cannot well be followed, and complex intellectual processes cannot be thoroughly performed."

A review of the history of this case, as thus imperfectly elicited, seems to indicate that, had the patient been under careful observation, a diagnosis of the prob-

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[1] Pepper's System of Medicine, vol. v., p. 89.

[2] Transactions of the Congress of American Physicians and Surgeons, vol. i., p. 272.

able seat of the growth would have been made, and, possibly, with sufficient certainty to have rendered an exploratory opening justifiable. Could the tumor have been located some months or a year before death, its removal would have been comparatively easy.

Dr. Ferrier, in commenting upon a case, very similar to the one reported in this paper, in regard to tumors in this situation, says:[3] "It is not until the tumor presses backward and, directly or indirectly, disturbs the functions of the central and posteriorly situated convolutions, that motor or sensory symptoms declare themselves; and until these have occurred, or until there are signs of implication of the nerves or other structures in the anterior fossa, an accurate diagnosis of the position of the tumor cannot be made with certainty, inasmuch as loss of memory and impaired power of concentration may also be produced by diffuse lesions or lesions which cause general disturbance of the brain. Of the structures in the anterior fossa, the olfactory tract is especially apt to suffer, but, on account of our patient's mental condition, it never could be ascertained with certainty whether there was any implication of smell in the one nostril or the other. During life, however, I had expressed the opinion that the case was in all probability one of tumor of the frontal region, but its precise position, whether deeply seated or superficial, or connected with the corpus callosum, did not appear to me sufficiently clear. For this reason, though I frequently considered the advisability of operation, I did not think it well to interfere until definite localizing symptoms should declare themselves; but the patient was carried off by an intercurrent malady before this event occurred. The necropsy, however, proved that the tumor might have been reached and, in all probability, enucleated by trephining over the left frontal lobe anteriorly."

DISCUSSION.

Dr. JAMES HENDRIE LLOYD.—About a year ago I put on record a case of tumor in the frontal lobe not exactly in this situation. That case had been successfully diagnosticated, and the tumor had been localized. A consultation was held to consider the propriety of trephining; but the man developed tuberculosis of the lung, to which he succumbed rapidly, and the operation was not done.

[3] Lancet, June 4, 1892.

In that case the most prominent symptom, and one which I have never seen in any other brain-lesion, was the peculiar inhibition of thought—the increase of the time-reaction of cerebration. Although Dr. Ferrier, in the communication referred to to-night, says that it may be present as a symptom of other lesions, I have never seen it in any other lesion so characteristic and so typical. In the case which I reported, the man was intelligent and answered questions properly; but it took him a long time to do so, as though, to use a simile, the question had to percolate through a stratum of diseased tissue. I have a suspicion that if there had been an opportunity to study the case reported to-night by Dr. Morrison, it would have been found to present these peculiar psychic symptoms.

Dr. CHARLES K. MILLS.—A similar case of tumor of the brain I reported years ago to the Pathological Society of this city. In the size and position of the tumor, the case reported to-night reminds me of it. It was a fibroma, involving the pre-frontal lobe, and extending to the corpus callosum and the gyrus fornicatus, but not to the motor region. The patient showed this slowing of the mental reaction. After shouting at the man, we would have to wait sometimes a minute for his answer. As I put it at that time, it seemed to take a long time for an idea to get into his brain or to be appropriated by his brain. He also had motor symptoms on the opposite side of his body, which, however, were unlike the symptoms seen in tumor of the motor region. He had either a pressure or inhibitory paralysis which was not absolutely persistent. Hughlings Jackson holds the view that the pre-frontal lobe is not only in a general sense concerned in higher mentation, but also in a certain sense it is a higher motor lobe. I believe that in such a case as that presented to-night, operation would almost certainly have resulted in death, unless great attention were paid to closing the dura mater and the scalp. One cause of death after such operations, as in porencephalus, is disturbance of the relation between external and internal pressure.

The PRESIDENT.—This tumor extended so very far back, that it is difficult to believe that there were no other symptoms than those recorded; yet when we look at the lateral surface of the brain, and see the marked difference in the course of the outer branch of the pre-central and also of the central fissures of either side, we see that

the pressure has been exerted very gradually. This is probably the reason that there was no weakness on one side. This fact has of course been illustrated by a great variety of brain tumors. The Society may remember that, some years ago, I presented a large mass, almost as large as this, which started in the base of the second and first frontal convolution, and which pressed back the motor area to a great extent; but there had been no marked motor symptoms, the parts evidently having adapted themselves perfectly to a gradually increasing pressure. The mass extended back so far that certainly the caudate nucleus and the anterior limb of the capsule must have suffered, and if the growth had been rapid, other parts would have suffered from the transmitted pressure.

Dr. WHARTON SINKLER.—This tumor is very much like one that Dr. Catell will remember, the specimen of which I presented to the American Neurological Society. This was a sarcoma occupying a space just in the Rolandic fissure. It grew downward from the dura, and by pressure caused complete right hemiplegia. The patient also had some convulsions.

Dr. JAMES HENDRIE LLOYD read a paper entitled

ARTHROPATHY IN GENERAL PARESIS.[4]

He first briefly traversed the history of the subject of the arthropathy of tabes and other organic nervous affections, referring to the pioneer contributions of Dr. John K. Mitchell in 1831, Scott Allison in 1846, and of Charcot in 1868, and the more recent contributions of Ball, Clifford Albert, Rosenthal, and others. The different varieties of arthropathies were fully considered, and the arthropathy of Charcot discussed with reference to its occurrence in four diseases—namely, locomotor ataxia, syringomyelia, disseminated sclerosis, and general paresis. In a note, Dr. Lloyd referred to one case of possible insular sclerosis with arthropathy which was operated upon by Dr. J. W. Hearn for the removal of numerous foreign bodies of bone from the knee-joint. He believed that, in general paresis with arthropathy, either posterior sclerosis was present or there was good cause to suspect it. He regarded the arthropathies as trophic lesions and

[4] Dr. Lloyd's paper, which systematically discusses the subject of arthropathies, will appear in full in the second volume of the Philadelphia Hospital Reports.

of spinal origin. The patient whose case was reported in detail, and which formed the basis of the contribution, belonged to Marie's hypertrophic type of arthropathy.

J. D., aged thirty-eight, white; admitted to the wards in July. He had suffered for some years with pain in the legs, which had been called rheumatic. Otherwise he was apparently healthy up to one year and a half ago, when he had a swelling of his knees. At this time no mental symptoms were observed. Six months before admission he began to have mental symptoms; he had delusions of a persecutory tinge. He became silly and weak. Then he had a remission and was much better; then he relapsed, and was sent to the hospital. Drawling speech began three months before his admission. He had had no convulsions. There was no history of syphilis.

On admission he was very demented and dull, answering questions very imperfectly. He had a drawling speeeh and some unsteadiness of the facial muscles. There was very slight nystagmus; no intention tremor; no delusions could be demonstrated at first; the knee-jerk appeared to be abolished, but the knees were so swelled and deformed that it was difficult to test them. By tapping vigorously, a slight myotatic reflex was caused in the quadriceps. Both knee-joints were enormously enlarged; grating sounds could be heard in them easily. The bony structure on the anterior end of the condyles of the femur was enlarged, forming prominent bosses. Sensory tests were negative, because of the patient's state. The pupils acted on accommodation, but did not react to light—the Argyle-Robertson phenomena. Hearing was good; the right elbow was deformed by an old ununited fracture of the olecranon, but the man had fairly good use of the joint; the detached olecranon was easily movable and somewhat enlarged. The left forearm presented an extensive scar, the result of an old injury; the ulnar nerve had been cut across many years before, and there was the characteristic wasting and *main en griffe*.

A few days after admission the patient had a maniacal episode—so characteristic of paresis—which required that he be strapped in bed. He was very violent in his delirium, fighting and resisting, but showing no intelligence. After this episode he gave evidence of a grandiose delusional state. He was more demented, and talked

in a rambling and incoherent way about finding an immense piece of silver and large sums of money. His speech became still more drawling and unsteady. Because of the deformity of his knees it could never be satisfactorily tested whether he swayed with his eyes shut. He managed to walk about. In the course of a few weeks incontinence of urine set in.

The joints grew more deformed. The patellæ were flattened and disfigured and increased in diameter. The bosses on the condyles were very prominent. No stalactites could be determined; grating sounds were always readily obtained; the joints were painless and free from heat or redness; there was evidently increase of fluid within them; no very marked œdema was seen around the joints, but still the skin had a very hard and tough feeling; there was increased lateral movement.

The electrical tests were of great interest. Very strong currents, both of faradism and galvanism, failed almost completely to cause contractions in the muscles of the thighs and legs. The only exception was in the peroneal group of the right leg, in which the reaction was normal. I think the reason for this failure was in the fact that the brawny and somewhat œdematous skin resisted the passage of the currents.

An examination of the eye grounds by Dr. Gould gave negative results.

Early in October the patient began to have retention of urine. This required the use of the catheter twice a day. Although every precaution was taken against sepsis, the urine soon became alkaline and contained pus. The bladder was washed out with a solution of boracic acid daily. On the 16th inst. the man had a severe chill, followed by a rapid rise in temperature. The chill was repeated daily. The patient's condition became grave. A diagnosis of infectious cystitis and pyelitis was made. He died on the 20th inst. with œdema of the lungs.

The post-mortem findings were as follows:

Brain.—The dura was very adherent to both the skull and the membranes. It was thickened. The longitudinal sinus was filled with a black clot. There was sub-arachnoid œdema. The frontal lobes especially were abnormal and in marked contrast to the rest of the brain. They were blanched, the arachnoid somewhat opaque, the convolutions evidently shrunken. The pia mater stripped easily from the brain in the Rolandic and parietal regions of the left hemisphere, but on the frontal

lobe it adhered, especially over the exterior and extreme frontal region and along the gyrus marginatus. On the right hemisphere this condition was more marked and more extensive. Almost all these convolutions tore when the pia was stripped from them. The perivascular sheaths were filled with a white opaque fluid. At the base of the brain adhesions were very extensive, so much so that it was difficult to open up the Sylvian fissure, the opposing surfaces being agglutinated together. The pia-arachnoid was thickened and opaque and strongly adherent in other regions of the base. The large blood-vessels at the base were normal. On laying open the brain-axis the lateral ventricles were found to be dilated and full of a clear fluid in excess. The ependyma was not thickened, but normal in appearance. The aqueduct of Sylvius was pervious. The walls of the third ventricle were agglutinated together. The basal ganglia, internal capsules, mid-brain, pons, medulla, and cerebellum were normal to naked eye inspection.

The Knee-Joints.[5]—The right joint was as follows: The synovic membrane in front was of a steel-gray color, mottled with blue patches and covered with numerous small miliary calcareous nodules. The anterior edges of the condyles of the femur were greatly hypertrophied in nodular masses or rogosities. These formed bosses extending upwards and outwards about two centimetres beyond the edge of the condyle. In the anterior notch between the condyles was a triangular mass of nodules embedded in tissue and freely movable. These nodules were of stone hardness. Over the articular surface of the right condyle there was left the lining membrane of the joint considerably thickened and rough in some places, in others thin, and presenting about the centre an eroded patch. Over the surface of the left condyle, the lining membrane was absent, and the bone was worn and eroded, especially toward the periphery. There was a series of small eroded patches with nodules running along the outer border to the posterior extremity of the condyle. In brief, most of the soft structures of the joint showed destructive changes. The semi-lunar cartilages were much worn and eroded, and easily displaced. The inner surface of the capsular ligament showed some pedunculated masses. The crucial ligaments were ap-

[5]Very accurate plaster casts were made of the joints by Dr. H. G. Cattell, Assistant Pathologist of the Hospital, and are now in the Museum.

parently wasted, but still held firmly. The end of the tibia was covered with thin, worn and eroded membrane; part of the surface of the bone was bare. The joint contained a green, thick, opaque fluid. The patella was quite deformed. It was thin and flattened, with rugous edges. The under surface was covered with shreds of membrane. The capsular ligament was much distended and the joint cavity extended to an abnormal limit upon the front of the femur. The left knee-joint was rather larger and more distended than the right. The same general characteristics were to be noted—distended capsule, eroded membranes, nodular pedunculated formations, and bony bosses on the anterior edges of the condyles.

Kidneys and Bladder.—The kidneys were large; the capsules adherent. The pelvis of the right kidney was enormously dilated and filled with a purulent fluid. The ureters were dilated. The bladder was thickened and presented evidence of a recent catarrhal process.[6]

DISCUSSION.

Dr. CHARLES K. MILLS.—This patient was in my wards also, and I had an opportunity of studying him a few weeks before death, and also just before death. It was certainly a most interesting case of arthropathy, and particularly on account of the paretic dementia which was present. As to this question of paretic dementia and the occurrence of arthropathies in the course of the cerebral affection, I think that one of the expressions which Dr. Lloyd used might be misleading. He spoke of these cases and other cases as belonging to the ascending type of posterior sclerosis. One of the earlier views of paretic dementia, when it was the termination clinically of posterior sclerosis, was that the degenerative conditions in the spinal cord continued through the motor tracts all the way to the cerebrum and to the cerebral cortex. This is certainly not the correct view. A number of years ago I had for several years a case of posterior sclerosis under my care in private practice. The patient became paretic and went to the Pennsylvania Hospital for the Insane, and subsequently to Danville, where he died. The body was sent to Philadelphia, and a post-mortem was made, and twenty or thirty sections from

[6] Acknowledgments are due to Dr. H. D. Beyea and Dr. Joseph Sailer, resident physicians, for valuable clinical work on this case. The specimens will be examined microscopically.

the cord, and all the way to the cortex, were examined under the microscope. Similar cases have been recorded. The disease perhaps ascends so far as the cord is concerned; but the cerebral condition is only a localized expression of a general condition. The disease does not usually extend anteriorly beyond the oblongata and pons. This has a little bearing upon the discussion of this case. We are not to regard these arthropathies as having anything to do with the paretic dementia. They are connected with the spinal lesion. The muscles were not extremely atrophied, and there is not much doubt that the absence of electrical reaction was due to the condition of the skin and subcutaneous tissues. Comparing the skin of the arm and hand with that of the thigh and leg, the difference was very marked; a solid mass, quarter to half an inch thick, seemed to be present under the skin of the thighs.

Dr. Lloyd refers to the occurrence of arthropathies in cases of hemiplegia, and in spinal affections other than posterior sclerosis. The arthropathies in cerebral hemiplegia are not to be explained in the same way as those of these cases. One explanation of them is that they are due to neuritis. They are due neither to the cerebral lesions, nor, strictly speaking, to the secondary changes in the motor tract, but to the peculiar conditions presented in hemiplegia, the relaxation of the joints and the traumatisms to which the limbs are subjected, and to the fact that the relaxation is itself in a certain sense a traumatism. As a result of the traumatisms inflicted by the flaccid, dangling, or sometimes contractured arm, neuritis is excited, involving a large part of the arm only, and sometimes the joints as well.

Dr. Lloyd referred to disseminated sclerosis with arthropathies. Of this I have seen one or two instances. In amyotrophic lateral sclerosis, I have seen peculiar painful and semi-anchylosed conditions of the joints, one within a few weeks, in the case of a lady who for three or four years has been developing symptoms of progressive muscular atrophy, or, more properly, of amyotrophic lateral sclerosis. Both shoulder-joints, but one more than the other, have been subject to attacks of pain without much swelling, but now there is a certain amount of adhesion.

Dr. WHARTON SINKLER.—I agree with Dr. Mills that the arthropathies of hemiplegia are quite different from those of spinal origin. Dr. Lloyd refers to the fact that

in some of the benign cases of arthropathies in tabes, recovery may take place in the joint. There is an interesting example of that in the wards of the Philadelphia Hospital. The patient has suffered from both epilepsy and locomotor ataxia, and at one time he had enormously enlarged knee-joints and was so ataxic that he could scarcely stand. I looked him up a short time ago, intending to show him in the clinic with some other cases of spinal arthropathies, but found that the knees had recovered and that he had regained the power of co-ordination to such an extent, that instead of walking with crutches he used only a cane.

I would like to refer to another patient at the Philadelphia Hospital who has remarkable spinal arthropathies and who is suffering from none of the forms of spinal disease referred to to-night. The patient has chronic myelitis, with contractures, exaggerated knee-jerk and trophic lesions. He had an enormous arthropathy of his shoulder, the joint at one time filled with fluid. Crepitation was also present. It was never painful. During the past year this swelling has very much subsided.

The PRESIDENT.—Some years ago I had a photograph made of the first patient to whom Dr. Sinkler has alluded. In addition to the enlargement, the knees were exceedingly retroflexed. There was no lateral displacement. Last winter when I wished to demonstrate the condition to my class, I found that while there was still some retroflexion, it had markedly diminished.

In the second case mentioned by Dr. Sinkler, the patient has a delusion that all his troubles are due to poisoning with arsenic. In this case there is loss of the temperature-sense over the entire right arm and side of the face, and it is probable that we have not a case of myelitis, but of syringomyelia with arthropathies in the shoulder-joints. In syringomyelia the arthropathies are more common in the upper extremity, while in locomotor ataxia they are more frequently met with in the lower extremity.

Dr. JAMES HENDRIE LLOYD.—I think that Dr. Mills may have misunderstood one point in my paper. I did not say that the joints in hemiplegia are like spinal arthropathies. I only referred to an historical fact. They were originally described by an Englishman, Scott Allison, in 1846, at which time the question came up as to their possible rheumatic origin. They are pseudo-rheumatic at least. They are painful and present some of

the symptoms seen in rheumatic joints. They are entirely distinct from spinal arthropathies. So the arthropathies of acute myelitis are apt to be of this same pseudo-rheumatic character, as in Gull's case and as in Dr. John K. Mitchell's case, which suggested to the latter his theory of the central origin of rheumatism. In 1831 he published a paper on this subject in the "American Journal of the Medical Sciences." These are different from the non-painless spinal arthropathies.

I have no doubt that in the case reported this evening, the connection of the joint disorder is with the cord, but it will require the microscopical examination to decide that. I do not care whether you call this case an example of the ascending type of general paresis or not. I have never believed that the degeneration in so-called ascending cases of general paresis begins in the cauda equina and extends upward. There is, however, sometimes a preponderance of posterior sclerosis in the earlier stages, which posterior sclerosis precedes the onset of the general paralysis. I have seen cases where the symptoms of locomotor ataxia existed for some years before the onset of the paretic dementia.

The PRESIDENT.—I might recall the fact that some years ago we had a collection of bones and joints exhibited to the Society. At that time I showed some bone sections. I found in some of the smaller joints of the foot that the cavities were obliterated and that bone tissue had been thrown across the joints. It is interesting, therefore, to note that the irregular deposit of bony tissue is not merely (at least in some instances) a calcareous deposit, but is made up of true bony tissue.

Dr. CHARLES W. BURR read a paper entitled

TUMOR OF RIGHT PREFRONTAL LOBE.

R. G., male, married, American, aged forty-four, box-maker, came to Dr. Sinkler's clinic at the Infirmary for Nervous Diseases, December 3, 1888, presenting the following history: During the summer he had suffered much from vertigo, thought to be due to dyspepsia. About August 1st, while standing in a street-car, his legs suddenly gave way beneath him and he fell. He was assisted home and put to bed, after which he became comatose and remained so the entire night. There was no convulsion. The next morning he was perfectly con-

scious, got up, dressed, complained of severe headache, and vomited several times. The third day he returned to his work and continued at it for three weeks. Six weeks later he had another severe attack, which was thought, by the physician who attended him, to be uræmic. On recovering consciousness, he saw double. He has not worked since. Diffuse headache and vertigo have continued. He has had one more attack of coma. Vision has been failing for several months. He uses alcohol very moderately. Syphilis is denied, and evidence of it cannot be found.

Present State.—Well built, healthy-looking man; gait good; station good; no ataxia in legs or arms; no paralysis; knee-jerks plus (later they were diminished); no impairment of taste or smell; hearing apparently normal, though no careful test was made; no pain on percussion of scalp; general sensation normal; mental condition good; no loss of memory; no slowing of mental activity; speech normal. Dr. Geo. E. De Schwinitz reported the eye condition as follows: "Discs swollen; R. E. + 6, numerous, tortuous, transverse veins pass over outer side. L. E. + 7 D. at apex; edges + 6 D. Irregularly contracted fields; lateral nystagmus." Urine free from albumen and sugar.

Further Course of Disease.—By February, 1889, there was complete blindness. From this time on the attacks of unconsciousness slowly increased in frequency, until finally two or more occurred every week. While his wife alleged that convulsive movements occurred in some of these attacks, this was never the case when I was present. The onset was always sudden; the coma complete; the respiration stertorious; the face congested; there was persistent and obstinate constipation. During the course of the disease the patient put on much fat. In the last six or eight weeks of life his mind became sluggish and he wandered slightly in speech. He died September 14, 1891, three years after the onset of the symptoms.

Remarks.—The headache, vertigo, attacks of coma, choke disc, and nystagmus rendered the diagnosis of intra-cranial tumor very easy. Its localization, however, was a matter of extreme difficulty, and the decision I finally made that the cerebellum was probably involved proved post-mortem to be false. I was largely influenced by the fact that in the absence of paritive localizing symptoms, nystagmus, with choke disc and complete

blindness, point to cerebellar disease. I explained the absence of cerebellar ataxia by non-involvement of the middle lobe. I think the error was excusable, or rather, the graver error was in attempting localization without sufficient data.

Autopsy was made nineteen hours after death, the body having been kept on ice. Rigor mortis was marked. The scalp was not adherent to the calvarium. The latter was very thin, and upon its inner surface were many worm-eaten depressions filled by small cauliflower-like excresences from the dura. The dura was congested, and over the right frontal region firmly attached to the bone and thickened. On removing the dura a softened mass attached to it came away from the outer aspect of the right prefrontal lobe. A harder mass was left behind, involving the entire lobe except the median cortex and the orbital surface. It did not extend into the ascending frontal convolution. Microscopically it presented the appearance of a round-celled sarcoma. In the softened part hemorrhage was present. The optic nerves were much shrunken.

SECONDARY CANCER OF THE DURA INVADING THE CEREBELLUM.

M. G., female, white, single, aged about forty-five years. In March, 1891, the left breast was amputated for carcinoma; and in April of this year the left axilla was opened, to remove cancerous glands. Immediately after this operation the left arm was found to be palsied. Soon after she began to complain of rapidly increasing weakness, and had an attack of unconsciousness without convulsion. There was at times slight frontal headache; never vomiting. I saw her once only, a couple of weeks before death, while she was under the care of Dr. Harvey Shoemaker, to whom I am indebted for permission to show the specimen. At that time she was emaciated, cachetic, and bed-ridden. There was no palsy of legs or arms except that due to the local injury; the lower face was palsied; tongue protruded straight; there was distinct inco-ordination of movement; the knee-jerks were much diminished; sensation was normal; there was constant lateral nystagmus; the pupils were equal, and reacted to light. Acuteness of vision could not be measured; she could, however, see apparently quite well. Dr. Cross reported that there was no fundus lesion; urine

was normal. She was apathetic, and almost never spoke except when addressed; she would answer all questions asked her, but soon became mentally fatigued, and then would reply at random. For some weeks before death she had attacks of sudden oncoming unconsciousness without convulsion; duration varied from one-half hour to several hours.

She died in June, 1892. Diagnosis: Intra-cranial cancer involving the cerebellum.

Autopsy.—A hard mass, about as large as an English walnut, was found growing from the dura mater at the posterior edge of the right lobe of the cerebellum. It had invaded the cerebellar tissue, and was surrounded by an area of softening involving the whole interior of the lobe. Microscopical examination showed it to be carcinomatous.

DISCUSSION.

Dr. WHARTON SINKLER.—This patient was a regular attendant of my clinic for nearly three years. At first, the diagnosis of brain tumor was readily made, but the difficulty in localizing the growth was exceedingly great. There were absolutely no focal symptoms. There was a history of a convulsion, but there was some doubt whether or not there had been any convulsive movements. In the subsequent attacks there was simply stupor. There was no pain, no headache, no loss of memory, no impairment of the special senses. No pain could be elicited by percussion. Therefore, from the standpoint of the neurologist as well as from the standpoint of the ophthalmologist, it seemed probable that the growth was located posteriorly.

The PRESIDENT.—It is exceedingly interesting to note that there were special attacks of stupor, which attacks seemed to replace the convulsions which are seen when the tumor involves the motor area. They seemed to be periodic attacks of irritation, causing, as their focal symptom, stupor. This certainly suggests a point for future use in diagnosis. If, as was claimed, the man really had a general convulsion, it was probably due to irritation of the dura, and not focal in character. The fact that the base of the second frontal gyri is pressed upon would be in harmony with the history of nystagmus. The case is also of interest as illustrating the difficulty of distinguishing between cerebral and cerebellar tumors. In a case at the Philadelphia Hospital, studied by Dr. Mills and

myself, it was impossible to make a positive diagnosis. There was general headache, and, as it was increased by percussion over the frontal region, we thought that the tumor might be in that position. Exploratory operation failed to reveal the growth, and the post-mortem demonstrated that it was in the cerebellum.

Dr. WHARTON SINKLER.—In the case referred to by Dr. Lloyd, the supposition of general paresis was justifiable from the circumstances. The man came into the house with distinct delusions of grandeur. He could walk a thousand miles; he had enormous strength; he had a large amount of money in his box at some sailors' boarding-house. These were the prominent symptoms on admission. Later, the unsteadiness of gait, pitching backward, and eye lesions were observed.

Dr. CHARLES K. MILLS.—One affection that should be borne in mind in considering the question of disease of the cerebellum, and that is labrynthine disease. The two affections sometimes resemble each other pretty closely. Of course, in labrynthine disease there is usually tinnitus and deafness, but you may have both in disease of the cerebellum. In labrynthine disease another symptom may be present, which is occasionally seen in cerebellar disease—that is, lost knee-jerk. I think that this may be explained in a method similar to that by which Jackson explains the absence of the knee-jerk in cerebellar disease itself.

Adjourned.

REMARKS ON THE VARIETIES OF CHRONIC CHOREA, AND A REPORT UPON TWO FAMILIES OF THE HEREDITARY FORM, WITH ONE AUTOPSY.[1]

By WILLIAM OSLER, M.D.,

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NOTHING illustrates so pointedly the widespread interest now taken in diseases of the nervous system than the rapid manner in which facts accumulate about obscure and rare affections. Twenty years have passed since Huntingdon, in a postscript to an every-day sort of article on chorea minor, sketched most graphically, in three or four paragraphs, the characters of a chronic and hereditary form which he, his father and grandfather had observed in Long Island. In the whole range of descriptive nosology there is not, to my knowledge, an instance in which a disease has been so accurately and fully delineated in so few words. No details were given; the original cases were not even (nor have they been) described,[2] but to Huntingdon's

[1] Read before the Philadelphia Neurological Society, Nov. 28, 1892.

[2] Several years ago I made an attempt to get information about the original family which the Huntingdons described, but their physician stated that, owing to extreme sensitiveness on the subject, the patients could not be seen.

account of the symptomatology no essential fact has been added. Within the past eight years a copious literature has gathered around the subject (particularly in this country), which is available to 1889 in the monograph of Huet.[3] Since this date the interest has even increased, and the references stand thick and close in the Index Medicus for the past three years. The recent paper by Sinkler (*Medical Record*, March 12, 1892) gives the literature to date. The practical outcome is that we now know the clinical aspects of this form thoroughly, and I have nothing unusual to offer in the history of two Maryland families which I have to report; but the connection of the chronic choreas with each other and their relation to chorea minor are questions which may be discussed, and upon which we need fuller information.

A chronic chorea of adults and aged was recognized long before Huntingdon's description of an hereditary form in adults, which was itself antedated in this country by the observations of Waters, Gorman, and Lyon.

Provisionally, at least, we may place the cases of chronic chorea in four groups:

First group, chorea of infants, appearing either at birth or within the first two or three years of life. Until recently but little attention has been paid to these interesting cases, of which there have been several well-marked examples at the Philadelphia Infirmary for Nervous Diseases; one has been reported by Sinkler and two by myself. A *résumé* of the literature to date is given by Audry in his recent monograph upon "Double Athetosis." The cases heretofore described may be, as he says, divided into those in which no accurate account is given as to the existence of spasm with the movements, and those with explicit statements as to its presence or absence. A majority of these cases are examples really of spastic diplegia, plus movements which may be choreiform, tremulous, or athetoid; or there may even

[3] De la chorée chronique, Paris, 1889.

be combinations of mobile spasm with more rapid movements, so that the diagnosis is extremely difficult, one observer calling the case chronic chorea, another double athetosis. This confusion was well illustrated in the discussion at the Berliner Gesellschaft f. Psychiatrie u. Nervenkrankheiten last year, when Remak showed a case of chronic chorea which Oppenheim had regarded as possibly athetosis, and which Senator thought—owing to the existence of spasm—had nothing whatever to do with chorea. In a large proportion of these cases there is also mental impairment, or even idiocy. The following case illustrates choreiform movements in a child with extremely slight spastic manifestations.

Female, aged four and a half years, seen in Ontario, May 12, 1892. One of twins, born prematurely at the eighth month. Mother had one child before, also at eighth month. Nothing abnormal was noticed about the baby at birth, it was not blue, and subsequently throve well. No abnormality was observed until the other child began to creep, when this one seemed backward and could not hold on to anything. At one year irregular movements were noticed in the arms and legs, and have continued. Teeth were cut at the twelfth month, and she began to talk at the third year; has never walked. The child is bright, intelligent-looking, with well-formed head; does not dribble. There is no nystagmus; talks a gibberish, of which I can only catch a word or two, but which the mother understands quite well. Movements slight in face, scarcely noticeable; no distinct grimaces; movements of tongue natural. The arms are in constant motion, slow and irregular as a rule, but occasionally jerky in trying to grasp objects. The fingers do not display athetoid movements. She cannot use a spoon, but can feed herself with bread, etc. The mother is sure that the arms are never stiff. She sits up well, but the head occasionally comes forward with a jerk. The feet are extended in talipes equinovarus position, and the toes spread occasionally in athetoid movements. The legs are freely movable, not apparently stiff; the muscles hard, but not very well developed. In taking off the stockings, however, the legs stiffened and were hard to bend at the knees, and the big toes became strongly flexed.

This case, belonging to the group described in literature as chorea spastica, is more properly a spastic paraplegia with choreiform and athetoid movements. The following is an illustration of a less common type, in which there was no spasm and the movements were of a more characteristic kind.

N. G., aged eight and a half years, the eldest of two children. The mother had twitching of the eyelids when young, but there are no nervous troubles of any moment in either her own or in her husband's families. The patient was a delicate infant, but throve fairly well, learned to walk and to talk at the usual time. About the fourth year it was noticed that she had irregular jerking movements in the arms, which were moved about wildly and even thrown over the head. She became excitable and irritable, and slept badly. Within a few months the face became affected, and she made grimaces, and sometimes a peculiar grunting noise. The legs were involved shortly after the face, and at times she walked with difficulty. When seen in 1890, more than four years after the onset, she seemed a well-grown child for her age, was not anæmic, a little nervous in her manner and excitable, but intelligent looking. After sitting quietly for a few moments, the arms jerked about and the face twitched. The right arm is most affected, and is twisted about in an odd way, and lifted as high as the shoulder. The legs are now not much, if at all, affected, though she fidgets about in her chair. When watched, the movements are much increased. She feeds herself with great difficulty. There is no spasm in the muscles, which are well nourished; the reflexes are not increased. There is no heart affection. Treatment has not been of the slightest benefit. She is very wayward; and though bright mentally, it is difficult to get her to attend to her studies. There have been no explosive utterances, or any of the mental features of convulsive tic.

And lastly, some of the cases of chronic progressive chorea with dementia have begun in early childhood.

Second group, comprising cases of chronic chorea without any hereditary *anlage*, in which the disease may set in in childhood, adolescence, maturity, or old age. Many of the cases in Huet's monograph had no history

of chorea in the ascendants. In scarcely any of the features are these cases to be distinguished from the variety described by Huntingdon, but in many instances the disease has begun in childhood or adolescence, and has gradually led, in a variable period of time, to dementia. Very many cases of this kind have been reported recently from asylums.

Only some of the cases of chorea in the aged can be classed here, since many run an acute course, and recovery is not uncommon, noted indeed in eleven instances in Herringham's critical review upon chronic chorea, in *Brain* (1888). The acute course, and the association occasionally with rheumatism, render it probable that many of these are really instances of chorea minor.

Third group, including the cases with marked heredity, the so-called Huntingdon's chorea, characterized by the occurrence in family groups, a late onset, psychical disturbances, and a progressive and fatal course.

Fourth group, comprising cases of chorea minor which pursue a chronic course, and persist for months or even years, and ultimately recover. They differ essentially from the other forms we have been considering, in the absence of a progressive character, the more active, quick, bizarre movements, and the retention of the mental powers. The following is a good illustration of the chronic form of chorea minor:

Alfonzo G., aged twenty-one, baker, applied to the Infirmary of Nervous Diseases, June 1, 1885, with spasmodic movements of the muscles of the face, arms, and trunk. The affection had lasted without intermission since August, 1884. There was no rheumatic history in the family, but a sister had chorea, and subsequently died of heart disease. He is a strongly built, well-nourished young man. The muscles of the head and neck and those of the face contract suddenly, jerking the head upward and rotating it slightly. At the same time he makes a grimace, and the muscles of the thorax are thrown into quick action, and the air is drawn in often with a whistling sound. The heart is not involved.

The patient was under observation and treatment for three years, during which time the chorea persisted with

slight variations in the intensity of the movements. When I last saw him the twitching and jerking of the muscles of the neck and chest were present, but the facial spasm had lessened. There were no mental symptoms, and for the greater portion of the period he was able to work.

Other instances of chronic chorea minor in the records of the Infirmary are given in my "Lectures on Chorea,"[4] two of which are very interesting from the persistence of the symptoms for more than three years with ultimate recovery.

Habit spasm, beginning in childhood, may persist for years, and is often confounded with chorea minor; there are also aggravated forms of convulsive tic with movements typically choreic, but which can usually be separated from chorea minor by the existence of fixed ideas, coprolalia, etc.

The following is a record of two family groups of the hereditary form of chronic chorea. The cases present the usual peculiarities described by Huntingdon. For the opportunity of seeing the members of the first family and for the details of the pedigree, I am indebted to Dr. Ellis:

FAMILY X.—FIRST GENERATION.

A. B., an Englishman, married C. D., a native of —— County, State of ——, and had of issue eleven children. A. B. died aged eighty-seven, and his wife aged eighty-five. Neither of them, so far as is known, displayed any mental or bodily peculiarities. Two of the eleven children died choreic and demented.

Of the other children, two of the girls married N.'s. One died aged seventy-five, leaving children, all of whom are in good health; the other, Mrs. N., still lives, aged seventy-seven, and has healthy children: George, died aged seventy, a bachelor; Sarah, died aged fifty, of typhoid fever, without issue; William, died aged seventy-six, leaving a large family, none of whom have shown any symptoms of the disease; Mary, died of an acute illness, aged fifty-five, leaving healthy issue; Jane, died

[4] Medical News, Philadelphia, October, 1887.

aged seventy, leaving a family, none of whom are affected; two other daughters died maidens, well advanced in life. The two affected children were James and Margaret.

James, the first to become affected, began to exhibit remarkable muscular irregularities before he was forty. Dr. Ellis writes: "I very well remember, in my earliest youth, his grotesque movements, exciting unusual attention, and I fear more ridicule than sympathy. His swaying, jerking, and fantastically irregular walk compelled him from the sidewalk to the unobstructed roadway. Notwithstanding his infirmity, he was a great pedestrian, frequently walking from his home, eight miles distant, and returning the same day. His sudden stops and precipitate advance, his facial contortions and mobile features, I recall with great vividness after forty years. His wife died in childbed.

Margaret, married J. M. Her symptoms began to develop before she was forty. She continued to go about until a few days before her death, which occurred in her sixty-fifth year. Except a short time before her death, she was not entirely helpless, nor were the mental symptoms very strongly marked in her case.

SECOND GENERATION.

Margaret M., the last-mentioned patient, had five children, two of whom have already died of the disease, and three are in various stages of it. I have seen two members of the family, and have performed a post-mortem on a third:

First child, male, now in his sixty-first year. A year ago the first evidences began. "A man of some character, it is but charity to ascribe the eccentricities of his life to disturbed mentality. He married twice, but had issue only by his first wife. Several children died in infancy, but one surviving is now in good health." This patient I could not see.

Second child, female, married, became choreic in her fortieth year, and died demented in December, 1890, in her fifty-eighth year. She was confined to her bed for nearly a year before her death, which occurred in the Pennsylvania Hospital for the Insane, Norristown. She had four children—three girls and one boy; all are living and in good health, the oldest being now in her thirty-second year.

Third child, male, aged fifty-five. I saw this patient with the doctor. He has enjoyed good health, and has been able to attend to his business until recently. When about forty-two he began to get nervous. Irregular locomotion was the first symptom; his speech became affected about a year ago. He will make use of a nod or a grunt in place of words whenever he can. Lately he has been confined to the house, and has been obliged to abandon business. He is very irritable, and is steadily passing into a state of dementia. He has had five children: four are living and in good health, the oldest about thirty-three years of age; one died of basilar meningitis at sixteen. I saw this patient in April, 1889, and made the following note:

Bony, well-built man; face has an intelligent expression. The gait is very peculiar; he sways from side to side; the movements are irregular, very unlike those of an ataxic, but resemble rather those of an alcoholic. He does not use a cane; feet are not specially spread; eyes not directed to the ground. He can stand with his heels together, with his eyes shut; no movements of the hands or arms when at rest, but in attempting to move there are large irregular sweeps of the arms and slight tremor. He has great difficulty in feeding himself, and sometimes takes two hours or more at a meal. He still can write, though with increasing difficulty. He signs his name to a letter, but the pen, in forming the letters, is often jerked up and the signature is very irregular. With the eyes shut he touches the nose or ear with precision and quickly. The grasp of the hand is firm and strong. There is no disturbance of sensation, no numbness or tingling. Knee-jerk slightly increased; ankle clonus not obtainable. Pupils medium size; react to light and on accommodation. Speech is slow, and interrupted frequently by the interjection 'Hem, ha!' This peculiarity, his wife says, is of comparatively recent development. The mental condition is apparently good; perception clear. When questioned, however, on several occasions, it seemed to take him some time to understand our wishes. He takes an interest in what is going on; reads a good deal, particularly the newspapers. He still personally conducts his business.

Within the three years and a half since making the preceding note he has steadily declined mentally and bodily.

Fourth child, female, aged forty-three, married, has had five chidren. One died of scarlet fever; the others

are living, the oldest a boy of twenty-three. In this case the disease has progressed with greater rapidity than in the others, and certain indications of it have been present, according to the doctor, since her thirty-fourth year. The mental symptoms were first to appear. In April, 1889, I made the following note:

Slightly built, somewhat anæmic woman; talks clearly and rapidly, but occasionally she displays a certain childishness, and the doctor, who has not seen her for some years, was much struck with the change in this respect.

While sitting quietly there were no irregular movements of her limbs, but occasionally there was a slight jerk of the finger, the shoulders would move, and once or twice, while speaking, there appeared to be irregular contraction of the facial muscles. There is no tremor of the tongue, and the pharyngeal muscles act normally; the grasp is good; she can use her fingers for delicate movements, and can thread a needle, and there does not appear to be the slightest inco-ordination. The most marked change appeared to be noticed in her gait. She walks with the feet somewhat spread, but follows a straight line fairly well; she turns with a little difficulty, and, if rapidly, loses her balance. Her head is carried somewhat stiffly in walking; she does not trip, and she walks in the dark quite well. She stands with her eyes shut and her feet together without swaying.

The power of the legs is good; knee-jerk increased on both sides; no disturbances of sensation; special senses normal; the pupils are of medium size and react to light and on accommodation. In the three years since the preceding note was made she has lost ground rapidly, and the muscular inco-ordination has become much worse. She is now confined to the house, and for the greater part of the time to her bed.

Fifth child, female, aged, at the time of her death, fifty-one; married; had eight children. Dr. Ellis writes: "After the birth of seventh child, in her thirty-second year, her husband noticed the beginning of the trouble in jerking movements of the legs when sitting, and when erect she had a trick of raising her heels suddenly and standing upon the ball of the toes. Irregular movements of the arms speedily followed. When I saw her first, in 1880, she could walk a mile or two without apparent fatigue, and would insist on walking to church, nearly a mile distant, repelling the suggestion that she could not walk as well as another. At this time, in walking, her

body would be bent forward, her head jerking, with a pendulum-like motion, to and fro, and her legs making such irregular and large movements that she would make wide excursions on the sidewalk. A year later she could no longer go out without assistance. Her speech indicated marked changes very early, in her fortieth year, and this was (in 1881 and 1882) accompanied by great difficulty in swallowing and frequently with alarming spells of strangling. She was a most pitiable sight. She suffered also from procidentia uteri; yet in June, 1883, in her forty-third year, she was delivered of her eighth child, which survived but a few days. Her menses were perfectly regular, her menopause occurring in her forty-eighth year. Six months before her death she was confined to her bed, utterly helpless, and was fed with a spoon. She was now entirely demented.

"Her deep reflexes were rather exaggerated. She could go about the house at night with as little help as in the daylight. She was exceedingly irritable and cross. The choreic movements stopped in sleep; there was no palsy of the sphincters. Of her eight children, seven are living, the oldest in her thirty-third year; all are in good health."

Post-Mortem (about thirty hours after death).—Considerable wasting of the body; no enlargements of joints; no abnormal position of limbs; face a great deal wasted, presenting several recent scars and abrasions, the result of falls.

The skull-cap of moderate thickness; dura tense; meningeal vessels looked stiff; longitudinal sinus contained recent clots. On the exposed cortex cerebri the arachnoid was somewhat turbid and universally separated from the pia by a considerable layer of serous exudate; this was especially marked over the sulci. Pacchionian granulations were numerous; cortical veins moderately full. At the base the arachnoid was turbid and the larger arteries a little stiff; the meninges were not especially adherent, and the pia could be stripped without tearing the substance. Superficial examination revealed no areas of softening, and no special lesions of hemispheres or of cerebellum. There was general wasting of the convolutions, which were also, on section, rather firm. The gray matter was dark, and in places looked thinner than normal. The crura presented no signs of descending degeneration; the pons and medulla were natural-looking; anterior pyramids had a clear, normal

aspect; the ventricles were not distended. Spinal cord was firm; arachnoid a little opaque; pia normal. Transverse sections showed no systemic degenerations; the gray matter had a rosy red tint.

Microscopical Examination.—I am indebted to Dr. Gray for an extensive series of sections from various parts of the brain and cord. The changes may thus be summarized: The arteries were thickened and in places showed hyaline degenerations, and, in the smaller arterioles, fatty changes, very marked in the fresh specimens from the cortex. Here and there the perivascular lymph-spaces were large and contained leucocytes. The ganglion cells in many sections showed very slight changes, not more than are often seen in chronic disorders associated with atrophy of the convolutions. There was the common vacuolation, and many cells seemed laden with pigment. The increase in the connective-tissue elements was more evident to the touch and on section than microscopically. Sections of the pons and medulla showed no special foci of disease. Beyond thickening of the arteries and a shrinkage in the cells of the anterior cornua (probably an artificial change), the sections of the cord showed no important lesions.

The morbid anatomy of chronic chorea is that of a neuro-degenerative disorder—diffuse changes in vascular, ganglionic, and neurogliar tissues—not essentially different to, though less pronounced than, those of dementia paralytica. We see, too, the terminal series of the process, far removed in time from, not necessarily akin to, the initial alteration which lies at the basis of the disordered function.

The doctor writes that, prior to the onset of the chorea, "these patients and their children are intelligent and bright, and the women are comely. The men are rather aggressive, energetic, and ferocious; the women are affectionate and prolific: the issue of the five numbers twenty-seven. There is no history of infantile chorea in the family, nor of rheumatism, nor of heart disease. The period of development of the symptoms covers a wide range, from the twenty-second to the sixtieth year. The symptoms have begun earlier in the women than in the men. There is at present no sign of disease in any

member of the third generation, though several of the children are past thirty-five. There seems to be a remarkable insensibility to pain in these cases; they fall about and bruise themselves severely without complaint. Shortly before the death of No. 4, she struck a cast-iron key, lodged in the door-lock, with her hand and broke it, naturally bruising and maiming her hand very much; but of this she took no notice whatever. The uncle and the mother of these patients kept about and showed much greater muscular vigor than members of the second generation, in whom, too, the dementia has apparently progressed more rapidly. The progress of the disease is marked by great emaciation; the movements are but little under control of the will and are much excited by volition. When standing, only those muscles are much affected which are concerned in balancing the spinal column and the head; the movements stop during sleep. These patients have all been light sleepers. The speech defect is not aphasic, but muscular —an indisposition to articulate on account of difficulty in moving the muscles. In case No. 4 the symptoms were very similar to those of a case of bulbar paralysis."

NEITER FAMILY.

So far as can be ascertained only four members of the family have been affected, namely: mother and three children, one of whom was our patient, Peter.

1. The mother, a German, is stated to have had trouble of the same kind as that which Peter has. For many years she made wild inco-ordinate movements with her arms, and toward the end of her life she could not eat alone and had to be fed. Her mind, also, became very weak. The exact duration of the disease in her case could not be obtained, but it extended over several years. She is said to have died of heart disease. She has one brother living, aged eighty-three, who is said to have the disease, but Dr. Simon visited him and reports that he is only subject to ordinary senile tremor. No information is available with reference to her family. Her maiden

name was Schmidt. She had four children, of whom three have been affected with the disease.

2. Lizzie N., was well up in her thirty-seventh year; married and had six children, of whom two died and four are living and well. After the birth of her last child the chorea developed, beginning in her arms first. Her husband noticed that she frequently dropped things. The trouble gradually became worse. Her mind became seriously affected, she talked incoherently, and had strange ideas. She once tried to commit suicide by jumping out of a window. The last year of her life she was helpless and could not walk alone. She died in her forty-ninth year, about twelve years after the first onset of the symptoms. Her husband, from whom these facts were obtained, says that the disease looked very much like St. Vitus' dance.

3. Nicholas Neiter, aged about forty, blacksmith, living at Edgewood, Hartford Co., Md. He was seen for me by Dr. Chas. Simon, who reports that he is evidently subject to the disease, as he displays grotesque inco-ordinate movements of the legs, arms, and face. Mentally, too, he is inclined to be childish and is very emotional. He regards himself, however, as in a condition of perfect health and not affected in any way as his brother Peter.

4. Peter Neiter, aged fifty-nine, German, a butcher, was admitted to Johns Hopkins Hospital,[5] October 9, 1890. Patient has been in this country since 1850. He has always enjoyed good health with the exception of malaria when he first came to this country; has not had syphilis. He dates his present trouble from an attack of gastro-intestinal disturbance eight years ago, which followed the drinking of large quantities of iced lemonade. At this time he had also pains in the head, and he speaks of the occurrence of something bursting in his body like a cannon. The movements began about five days after this over-heating and taking iced drinks. They did not start at any particular part of the body, but were general from the outset. They have gradually become worse, particularly when voluntary movements are made. They are severe enough to prevent him from working, and he has not been able to do much for six or eight years. He has fallen, sometimes, owing to the irregular movements of the legs. He has never at any time lost consciousness.

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[5] The patient was shown at the Hospital Medical Society, and is reported in the Bulletin, vol. i.

Emotion or fright always exaggerates the movements. He has not had headaches; has as a rule slept well. His appetite has been good and general health excellent. Ever since the attack, eight years ago, he has been liable to a recurrence of the vomiting whenever he takes cold drinks. He says his memory is quite good. He does not think that his speech has been affected.

Present Condition.—The patient is a large, well-nourished, well-built man. The face in repose looks intelligent, but on smiling, the expression is fatuous. He answers all questions readily and freely; gives a good account of his condition, and it is more in his expression and general behaviour that an indication is found of mental impairment.

When sitting in a chair, at ease, the arms and hands are in more or less constant irregular motion. The fingers are extended and flexed alternately; sometimes only one, sometimes the entire set. At other times the whole hand will be lifted or there are constant movements of pronation or of supination. For half a minute or so they may be perfectly motionless. The head and trunk present occasional slow movements; in the latter more of a swaying character. The legs jerk irregularly and the feet are flexed or extended; but the movements are not so frequent as in the arms. The face in repose is usually motionless, but the lips are occasionally brought together more tightly and the chin elevated or depressed. There is an occasional movement of the zygomatic and of the frontal muscles. He puts out the tongue, with tolerably active associated movements of the face, and it is usually quickly withdrawn or rolled from side to side. It is impossible for him to hold it out for any length of time. There are no irregular movements of the palate muscles.

He walks with a curious irregular gait, displaying distinct inco-ordination, swaying as he goes, hesitating a moment in a step, keeping the arms out from the body and in constant motion. The legs are spread wide apart; steps are unequal in length and he seems rather to drag the feet. He stands well with the heels close together.

There is a suggestion of stiffness about the gait and about the way in which he uses his legs.

Sensation is unaffected. The deep reflexes are increased. There is slight ankle clonus, exaggerated knee-jerk, and slight increase in arm-reflexes.

The special senses are unimpaired. Pupils are of

medium size—the right a little larger than the left; they react to light and on accommodation; there is no nystagmus. He has no fever; bowels are regular, and the urine shows no special changes.

A report of cases of the hereditary form of chorea does not afford a very wide scope for discussion; but there are problems in the relation of the forms to each other and to chorea minor, which, if I have read the literature aright, are still far from settled. My own point of view may be very briefly stated: Chronic progressive chorea is a malady distinct from the various disorders associated with coarse lesions of the motor centres or path known as symptomatic chorea—an affection which (like forms of muscular atrophy) may occur in families or in single individuals, and is characterized by irregular, inco-ordinate movements, a reeling gait, speech disturbances, and progressive impairment of the mental faculties. The movements differ from those seen in chorea minor, being slower, and resembling rather those of Friedreich's ataxia, without the brusque, jerky character of the former disease. Moreover, in striking contrast to the movements of chorea minor, those of chronic progressive chorea are sometimes influenced by the will. A certain number of the cases of chronic chorea beginning in infancy and childhood belong to this category, but a very much larger number are instances of spastic paraplegia or diplegia; while others represent anomalous forms of chorea minor.

Chronic progressive chorea is, I believe, a disease wholly apart from the affection described by Sydenham, having nothing in common with it but the name. The course of acute chorea minor, the incidence in children, the arthritis, the seasonal relations, the extraordinary frequency of endocarditis—to say nothing of the different characters of the movements above referred to—separate it as a well-defined affection, depending possibly on a virus as yet unknown.

Stated Meeting, November 28, 1892.

The President, Dr. FRANCIS X. DERCUM, in the chair.

Dr. WM. OSLER read a paper on

CHRONIC CHOREA. (See page 23.)

DISCUSSION.

Dr. CHARLES K. MILLS.—My attention has long been directed to the importance of finding the link which binds these cases of chronic chorea, not only to each other, but also to other well-known forms of organic and mobile disease, as Friedreich's ataxia and various organic athetoid and choreic diseases of that kind. I should like to know more about the exact pathology of these cases, and I had hoped that the microscopical examination which Dr. Osler had made would have thrown some light upon this. Certainly the so-called Huntingdon's chorea is a disease apart from other choreas, but probably not apart from some other forms of organic motor disease with which we are familiar, particularly in an institution like the Philadelphia Hospital. These cases are not infectious as the minor choreas may be, but rather they are due to some deficiency in the development of the nervous system, although it is curious that they should come on so late in life; but the same is true of some forms of sclerosis.

Dr. JAMES HENDRIE LLOYD.—There seems to me to be a well-marked distinction between chronic chorea of adult life and the acute chorea minor of childhood. I have always believed that they were distinct, depending upon a distinct pathology. The most interesting point in the consideration of chorea minor is the possibility of it being an infectious disease. While nowadays there may be a tendency to go to extremes in regard to microbes and bacteria, yet chorea minor presents a well-marked group of lesions, such as joint involvement and endocardial change, which suggest the probability of infection.

Another important point is with reference to the relationship of chorea to pregnancy or of pregnancy to chorea. This is another of the unsolved problems of chorea. I have myself had rather an unusual experience, having seen five or six cases of chorea of pregnancy. It is well known that some of these cases are very acute and rapid in their progress, and that others of them have a definite tendency to become chronic or to end in death, and others tend toward involvement of the brain and to insanity. I remember an interesting case in the Pennsylvania Hospital for the Insane, in which an ordinary chorea of pregnancy ran into a dementia which was hopeless, the choreiform movements continuing. The chorea of pregnancy offers some objections to the theory of the infectious origin of chorea. On the other hand, there are some things about it which suggest a moral relation or a mental impression. A large proportion of the cases of chorea of pregnancy occur in primipara, and especially in those illegitimately pregnant. Dr. Levick recorded, years ago, a most interesting case of this kind at the Pennsylvania Hospital.

These are unsettled problems. The chronic chorea of which Dr. Osler speaks seems to me to be distinct from these acute forms and probably dependent upon some degenerative central disease.

Dr. GUY HINSDALE.—I had hoped that Dr. Osler would have told us something about the treatment. I presume that when the disease is divided into several classes, the treatment applicable to one class would not be suitable for the others. While arsenic may be beneficial in Sydenham's chorea, it may not be applicable to cases occurring in adult life. A week or two ago I saw, in consultation, a case of chorea in a woman eighty-twa years of age. The disease had existed for six months. Arsenic and other remedies had been tried, and I was asked what to do; but I was unable to afford much encouragement in the use of further medication.

The PRESIDENT.—Dr. Osler's paper has impressed me more than ever with the fact that chorea is a symptom, not a disease; that is, we have a large group of choreiform diseases, many of which are essentially different. The ordinary Sydenham chorea and adult chorea are different diseases, as I think we are all willing to admit. The choreas of adult life present some interesting problems. With regard to spasm in these cases, my impression is that this does not occur in the hereditary forms of adult chorea.

I would ask Dr. Osler whether, from his experience, there would be any possibility of distinguishing, without the history, the cases of hereditary adult chorea from those which are not hereditary. According to my own experience, this distinction is not possible.

Dr. G. BETTON MASSEY.—Nothing has been said about alcoholism as a possible cause of this form of adult chorea. I do not know that there is anything in the idea, but the general impression among the commnnity is that in these cases the father or mother has been intoxicated at the time of conception, or that the disease is the result of maternal impression. I recently saw a young man, twenty-two years of age, who had been intoxicated on two occasions. He stated that after the first drink of liquor he was unable to withstand it, and three or four ordinary drinks of whiskey would cause sudden intoxication. In this case there was a family history of hereditary chorea.

Dr. WILLIAM OSLER.—I can answer the question about alcoholism so far as the families reported are concerned; none of the members have been heavy drinkers. If alcohol was in any way a factor in the production of chronic progressive chorea, the disease would be exceedingly common, whereas it is extremely uncommon.

The point raised with reference to the affinities of this disease is very important; for instance, its relation to double athetosis. Here the chief element of distinction in many cases is the spastic state of the muscles in athetosis; but in the discussion in the Berlin Society, to which I have referred, an interesting difference of opinion between experts was shown, one regarding a case as double athetosis, another as hereditary chorea, while a third thought that as spasm was present, it was probably not chorea. In the majority of the cases with spasm, I think the disease does not belong to this group although they are closely allied, and there may co-exist an athetoid with a choreiform condition. I do not think that spasm characterizes the non-hereditary form of chronic progressive chorea. So far as I know there is no essential distinction between these two forms other than the heredity.

The question of Dr. Hinsdale is interesting, but with our ample experience of the course pursued, no one should attempt to treat medicinally a case of chronic chorea. A physician should have the courage in these cases of chronic progressive affections of the nervous system, to say to the patient: "My good fellow, there

is nothing to be done for you. Go home, arrange your affairs, live a quiet life, and do not throw away your money on medicine, particularly quack medicine." Unfortunately chronic chorea contributes only a small part of the cases of chronic disease of the nervous system for which we can do very little. This is regarded as a great reproach to medicine, we cannot be expected to cure all cases; as old Sir Thomas Browne said two and a half centuries ago: "There are vices incorrigible in divinity, cases indissoluble in law, and diseases incurable in physic," and they will remain so.

Adjourned.

GUNSHOT WOUND OF THE BRAIN, CAUSING A FORM OF APHASIA IN WHICH THE LOSS OF NOUNS WAS THE STRIKING FEATURE.

By ERNEST LAPLACE, M.D.

Professor of Surgery, Pathology, and Clinical Surgery in the Medico-Chirurgica College, Philadelphia.

AUGUST 4, 1892, Lizzie Albert, aged nineteen, received a gunshot wound in the inter-ocular region, at the apex of the glabella; there was a fracture of the skull, and penetration of the bullet, according to the testimony of the physician who saw her immediately after the accident. She became unconscious and remained so during three weeks. Meanwhile, several spicula of bone were removed from the seat of the penetration of the bullet. The wound gradually healed, leaving a slight depression. As she recovered consciousness, she complained of a severe pain in the left occipital region, ranging downwards, constant and almost unbearable, keeping her mind always fixed upon that point; suffers no pain otherwise about the cranium. The patient is a well-nourished, strong young woman, showing no evidence of any diathesis or cachexia. As a result of the injury, most interesting intellectual symptoms resulted, her physical condition, however, remaining entirely unimpaired. When complaining of the pain back of her head, she says that she "sees it;" when asked "What?" she says she "sees it;" if asked, "Does that hurt?" she answers, "Yes." If asked, "What hurts?" she says, "It hurts." When asked, "Does the bullet hurt?" she says, 'Yes."

It seems that other organs of sense refer their impression to the same centres. For instance, if asked whether she hears a particular sound, such as a tuning-

fork, she will answer, "Yes, I see it." If given anything to take, and asked whether she takes it, she answers, "Yes, I see it." Likewise, if anything is given to her to smell, she also sees the sensation of smell. It seems to her an impossibility to repeat a word spoken to her, though she understands fully everything. In her conversation she uses no names of persons or things; she knows every person and everything. For instance, if shown a hat, and asked what that is, she will say, "It is yes;" but asked the name of it, she will say, "I do not know." If asked, "Is this a book?" she will say, "No." "Is it a hat?" She will say, "Yes." Should we write on paper the word cat, in presenting her the hat, she will say, "That is not it." If, changing the word slightly, we make it coat, showing her the hat, she will say, "That is not it." If changing the word slightly, and writing the word hat, and asked whether that was it, she will say, "Yes." If asked her name, she will answer, "I do not know." If asked whether her name is Katie, she will say, "No." If asked whether it is Lizzie, she will answer, "Yes." Asking her to write her name, although she says she does not know her name, she will write it correctly—Lizzie Albert. If asked whether that is her name, she will say, "Yes." If asked what her name is, "I do not know; I cannot tell you." Should I write her name, misspelling it in any manner, she will say immediately, "That is wrong." If asked, "What is wrong?" She will point out the letter in the word which makes it misspelled, and until the name be properly spelled, she will insist on its being wrong.

Her judgment seems to be perfectly clear; that is, she says or does nothing which would in the least compromise the condition of her intelligence. There seems to be a destruction of such fibres as lead to those portions of the frontal lobe as are the centres of memory for names of persons and things.

The lady, at whose house the patient has dwelt for the last two years, has since the injury exercised continued care over her, and at no time has the patient

shown violence or other signs of dementia. While the patient insisted on having such pain in the left occipital region, and while she pleaded for an operation to remove the bullet which she felt sure was there located, but little reason existed for us to pursue any other course, and we therefore thought best to explore the brain in that neighborhood, at no time having had much hope of finding the ball in that locality; consequently, on November 4, having observed the strictest precaution for the aseptic operation, the trephine was applied over the seat of the pain, on a level with the occipital ridge; the dura mater was found healthy, rather more congested than usual. A rongeur forceps was applied and a strip of bone, an inch and a half long and three-eighths of an inch wide, was removed in a perpendicular direction, parallel with the squamous suture. A malleable wire instrument, specially devised for this occasion, was introduced, and with it the separation of the dura mater was effected to a distance of two inches all about the opening, so that an area of the brain four inches wide and six inches long was explored. A normal condition was revealed everywhere, giving us absolutely no information as to the locality of the bullet. The bone was replaced, and the wound sutured and drained at the most dependent portion. At no time after the operation did the temperature rise above 99°. The patient recovered from the ether perfectly, and the subsequent treatment was uneventful. Ten days after the operation, all dressings were removed, perfect union by first intention, and the patient resumes her occupations about the ward. A certain change, however, is already noticeable about the brain symptoms, viz., consisting in a more talkative disposition and the ability to speak the names of two or three objects, such as: home, sister, country. As far as pain is concerned, the patient seems to be much relieved, and now feels assured that the bullet is situated near the seat of entrance, and begs for another operation for its removal.

Stated Meeting, December 19, 1893.

The President, Dr. FRANCIS X. DERCUM, in the chair.

Dr. ERNEST LAPLACE read a paper on

A CASE OF GUNSHOT WOUND OF THE HEAD, WITH A FORM OF APHASIA IN WHICH THE LOSS OF NOUNS IS THE MOST MARKED FEATURES. (See page 42.)

DISCUSSION OF DR. ERNEST LAPLACE'S CASE OF LOSS OF NOUNS.

By CHARLES K. MILLS, M. D.—In Dr. Laplace's case one of the chief features is the loss by the patient of the ability to make use of nouns, or at least of many nouns. At first sight the case might seem to be hysterical, but a close study of the phenomena, and the long persistence of a consistent symptomatology, are strong arguments against this assumption. The case can be explained in accordance with recorded facts, and on certain well-based theoretical considerations. The literature of aphasia contains a number of anthenticated cases—a few of which have gone the rounds of numerous publications—in which either the memory of nouns, or the faculty of using nouns in voluntary speech was lost. In 1887, Dr. Mary Putnam Jacobi, in a paper on *The Special Liability to Loss of Nouns in Aphasia*, published in the Journal of Nervous and Mental Diseases, as the results of the examination of the records of 116 cases, stated that 17 had this peculiar defect. These cases, most of which are well known to neurologists, are given in abstract. A few other cases are to be found in the medical literature of the last five year.

In many cases of aphasia the diminution or loss of facility and expertness in the us of nouns is a marked feature, but is associated usually with other striking disturbances of speech.

This peculiar liability to the loss of nouns has been discussed by our most eminent authorities on the subject of aphasia such as by Bouillaud, Broadbent, Kussmaul, Bateman, Ross, Jackson and Starr. It brings up an old question as to which parts of speech were first used, nouns or verbs, and the probable order in which the different parts of speech were added in the evolution of language.

The word-hearing centre is located in the posterior portions of the first and second temporal convolutions. The centre for word-vision is in all probability in the anterior part of the lateral aspect of the occipital lobe. The posterior part of the third frontal convolution is concerned with the arrangement of words and ideas, and close by is the convolution for the emission of speech. The existence of a separate area for concepts, or a "naming centre," as Broadbent has called it, is disputed, and this case is much to the point in discussing this question. Can we explain such a case in any other way than on the supposition of such a separate area? It seems to be difficult. The explanation of Ross is after all a practical admission of the existence of such a separate area. He says that it is not separate and distinct centre in the human brain, but that there is a higher organization or an expansion of the centres for word-hearing and word-vision; in the process of evolution the centre for word-hearing becomes enlarged and more highly organized; and similarly the visual area pushes outward and becomes larger and more developed, but, according to him, they are still parts of the same areas. We often become cognizant of an object and its name by the convergence of impressions from different centres, and the most rational supposition is that in process of time these pushings out from the original percept areas have become distinctly organized as a special intermediate area. Of two or three ways of explaining this case, one is by a destruction of the region where different lines converge to a common naming area.

A lesion of the lines of communication which go from this concept or naming region to the propositionizing area in Broca's convolution might partly bring about such a result. Although the patient would conceive the name of any object, he would be unable to carry the conception over to the emissive side of the brain because of the blocking of the connecting channels. An isolated

lesion of the naming centre would cause a loss of memory of names; the patient would not recall in consciousness the names of the objects; but as the patient is able to hear the word, to see the word and to recognize it, it would seem rather that the lesion has destroyed the fibres between the naming area and the out-going centres, whatever else it has destroyed. That any one is able to copy a word that is set before her is in favor of the view of a separate concept area. In two ways impressions from the visual area can go to the propositionizing area in the brain; one is direct the other indirect by way of the concept centre. Suppose that the indirect route is destroyed and that the direct commissure is intact; this would explain the fact that she can copy; and if she could repeat from hearing words, then also the direct route from the auditory centre to the out-going centre would be intact; but she cannot do this, so it would seem in this case on the supposition of separate concept centres, that the lesion has destroyed not only the indirect, but also the direct route from the auditory area, and has left the direct route from the visual centre intact.

That which is destroyed by such a lesion as this, is power of naming a tangible object represented by a noun. The nouns representing abstract qualities like home, liberty, country, love, hope, etc., are not necessarily, and commonly are not incapable of being used in cases like this, because a mental picture the result of visual or other perceptions does not rise into consciousness when we use these nouns representing abstract qualities.

Personally I believe in the existence of a separate naming centre, or at least of an area peculiarly organized, and lying between the centres for percepts and for motor speech—I believe, with Broadbent, that impressions made by an object upon various perceptive centres, as, for instance, those of hearing, sight, and touch, converge to one common cell, zone or area in which a complex impression of the object, the idea or the mental picture of the object, looms into consciousness. If such an area exists it must be placed in such a position as to be in easy relation with the percept centres, and also intermediate between these and the outgoing centres for speech in the third frontal and adjoining convolutions.

This brings up the most interesting practical point in connection with a case like that presented by Dr.

Laplace, in which surgical procedure was tried, and in which the advisability of further operation is still under consideration, and that is as to the direction and location of the lesion producing this peculiar variety of aphasia. In one case of loss of nouns reported by Broadbent, the two posterior convolutions of the island of Reil had disappeared. The island of Reil or retro-insular convolutions, or perhaps the inferior parietal convolution, best fulfil the anatomical requirements. It seems to me probable that either directly by the bullet, or, more probably indirectly, by intra-cranial traumatism or hemorrhage the left insular lobe has been injured

Let me summarize and to some extent repeat. This patient recognizes what is said to her whether nouns or other parts of speech. She can read silently, understanding what she reads. She can copy silently understanding what she copies. She can speak a little spontaneously, and more in this way than she can from dictation. She can propositionize imperfectly without nouns of a certain kind. She cannot name objects the use and meaning of which she knows and of which she probably knows the name; she cannot write the same. The auditory centre and incoming auditory tract are intact—so is the incoming visual tract and visual centre. The concept or naming centre may be partly preserved, because she has the idea of the thing, and she has the idea of the name. At first it was puzzling to understand why she did not use periphrase. Of the seventeen cases reported by Dr. Jacobi, while some of them periphrased very well, the majority did it poorly. In other words, the propositionizing power is impaired in a large number of these cases, and in some it is lost entirely The commissure between the naming and propositionizing areas is destroyed. She can enunciate words because Broca's centre is intact, but she cannot repeat from dictation. The direct route from the auditory centre to the convolution of Broca, as well as the indirect route to the naming centre is fractured. She cannot read aloud or will not; this is difficult to explain, but a possible explanation is that when we read aloud we verify what we are reading in consciousness by the auditory centre. It is well known that a communication exists between the visual area and the auditory area. She is unable to verify by her auditory centre that which she is able to pronounce through the motor cortex. The explanation is the same as that for many aphasics who talk jargon or gibberish. The lesion

is then probably one destroying both the association fibres from the concept to the motor speech regions, and those which go directly from the auditory centre to the motor speech area; but it does not directly involve the latter.

In this connection I would like to report without comment a case of Amnesia for names following an Operation on the left Gasserian Ganglion.

The patient, a man seventy-six years of age, had during the past six years undergone five operations for the relief of trifacial neuralgia of the left side, the present one being the sixth.

On November 19th, by Dr. John B. Roberts, a horse-shoe shaped opening about three and a half to four inches in extent, was chiseled into the side of his skull for the purpose of removing his gasserian ganglion; much pressure was made upon the brain to displace it in order to reach the ganglion. The second and third division of the fifth nerve were cut near to the ganglion and turned back into their respective foramina, the ganglion itself was not removed. On the second day after the operation it was noticed that the patient could not remember the name of the street he lived on, nor that of any other street in the neighborhood of his house; he could not remember the name of the hospital, nor of his doctors who had previously operated upon him. It took him quite a little while to remember the name of President Harrison and Cleveland, though he had been much interested in the political campaign. His memory of names gradually returned to him so that two weeks after the operation he knew the names of the streets etc., as well as he ever knew them.

The PRESIDENT.—I saw this case some time ago with Dr. Laplace but since then its character has changed considerably. At that time, the aphasia was greater than at present. It was in fact a complete or almost complete motor aphasia. The case struck me at that time as one of aphasis from concussion or psychic shock. We know that such aphasias actually occur and may be persistent. Of course the fact (?) that the case is a pure case of loss of noun memory, militates against this view, although not absolutely, for cases are on record where loss of memory of nouns has followed shock and there has been no reason to suspect special lesions. Whether in such cases there might be a molecular disturbance of a certain portion of the cortex which might be persistent,

is a matter for speculation. On the other hand, looking at the case as an organic one, a clot may have formed and the change in the case would be accounted for by the partial absorption of the clot. I do not think that we can say absolutely whether or not there has been hemorrhage.

It is difficult for me to understand why she should be able to write certain names while she cannot write or pronounce from a book simple substantives.

With regard to the existence of an ideation centre (as I prefer to call it rather than a naming centre), the familiar diagram of Lichtheim answers very well to illustrate it. We have an impulse, say passing up the auditory nerve to the cortical centre for hearing and conducted by fibres beneath the Island of Reil to the third frontal convolution. Here the sound can simply be repeated, or emitted. No act of ideation is required in such a piece of apparatus. Every one of us can read sentences in a foreign language which we do not understand. Other portions of the cortex must be excited in order that sounds heard may be translated into ideas.

One peculiar feature about the case is that she does not attempt to periphrase in order to avoid the use of nouns. If she has a memory of verbs, she should be able to say what is done with certain objects. We might explain her inability to read from a book in this way—her power for repeating nouns is gone, but the power for repeating verbs is present though impaired. If she *hears* a verb repeated the motor speech centre is much more stimulated than when she simply *sees* the printed symbols. If the case be one of concussion it is strange that it is so purely a loss of noun memory as it is; if it be organic, it is strange that it presents the peculiarities that it does.

We should remember the fact that this girl was shot under circumstances of the most profound emotional excitement. When I first saw her, the condition suggested hysteria rather than any serious lesion. The aphasia was apparently complete except for a few words, such as yes and no. There was no palsy nor any other symptoms. It looked like a psychic case and this view would be favored by the partial recovery. Whether or not hysteria would explain this case, is a subject open to a good deal of discussion.

Dr. WHARTON SINKLER.—One explanation of the ability to write Dr. Keen's name would be that of visual

impression. The patient had read the name just before she was asked to write it, and may have had a mental picture of it before her.

There are several facts that make it difficult to accept the theory of an organic lesion in this case. In the first place if the bullet is located, as is very probable, within two inches of the point of the entrance, how can we have a lesion so far back that it can affect the speech centre? Moreover, could the bullet penetrate the brain to the speech centre, without producing some other localizing symptoms? In the second place, I do not feel sure that we have established the facts as to whether she could not or would not read and write names.

Adjourned.

THE CHOREIC MOVEMENT.

By HORATIO C. WOOD. M.D.

IN A RESEARCH published by myself in the *Therapeutic Gazette*, in 1885, the observations of Chauvet, Carviler and several later investigators, that section of the spinal cord does not arrest, either in the upper or lower segment of the body, the choreic movements sometimes seen in diseased dogs, was confirmed by numerous experiments. It was further shown that whilst in advanced cases of canine chorea the ganglionic cells of the cord undergo distinct change, in the earlier stages of the disease, although the choreic movements may be well pronounced, no structural alterations can be detected in the nervous system. I also pointed out the close resemblance which exists between the chorea of the dog and the St. Vitus's dance of the child; and that since it is certain that the canine choreic movements originate in the spinal cord, it is most probable that the choreic movements in the human individual are often spinal. Since my research the matter has been considerably discussed by physiological clinicians, and it seems to me that the conclusion can, at this day, hardly be gainsaid, that whilst the movements which are called choreic may originate in the

cerebral cortex, spinal choreic movements occur in man as they do in other mammals. The assertion which has been made, that the mechanism of the choeric movement is altogether different in man and in the lower animal: that is, that it is altogether different in different species of animals, is too absurd for serious controversy. It would be just as well to waste time in discussing whether the mechanism by which voluntary movement is produced, or the method by which convulsions are brought about, differs in man and in the lower animals.

In the research of 1885, no direct answer was attempted to the question, "What is the immediate method in which choreic movements are produced;" or, in other words, why does the choreic movement replace the normal function of the part. It seems to me possible that the veterinarian and the human physician will agree that usually and probably always the spinal choreic movement is associated with absolute loss of power; a loss of power which varies in degree from mere failure of endurance to almost a complete paralysis.

The problem which is presented to us for study is the examination of the conjoint excesses of motion and loss of motor power. In thinking over this problem, it occurred to me that probably in chorea there is a condition of depression or exhaustion involving the two motor functions of the spinal cord, which I believe have been proven by the modern physiologist to exist, namely, the function of inhibition of motor discharge and the function of motor discharge. In other words, that the motor power of the spinal cells is weakened, but that the power of the inhibition apparatus, which controls motor discharge from these cells, is weakened to a greater extent than is the discharge power. The choreic movement may show itself simply in restlessness or fidgetyness, or it may reveal itself by excessive muscular contractions occurring under the impulse of the will; or in the severer cases there may be an irregular or a rythmic motor discharge, occurring entirely independently of the action of the will. According to the thought which I have, the quiet individual

is one whose inhibitory spinal apparatus is so powerful as to effectually control the motor cells of the cord; whilst in the restless individual inhibitory weakness reveals itself in the restlessness. As inhibitory weakness increases, restlessness increases, and the child becomes fidgety. When the stage of pronounced awkwardness and irregularity of movement is reached, the inhibitory spinal apparatus has so far lost control that it is no longer able to arrest muscular movements at the time when the necessities of the occasion demand that the motor discharge should cease, and so an excessive muscular response occurs to the will impulse because of the weakness of the apparatus which checks discharge from the cells concerned. In the most profound case of spinal chorea, human or animal, according to my thought, inhibition has become so far weakened that it has little or no power over the motor cells of the cord. As a further extension of the theory, the thought has occurred to me that it may be possible for a cell to revert to its original physiological type, just as it is possible for a part to revert to its original anatomical type. The original type of the nerve cell is that in which the cell gives rise to intermittent discharges, and therefore it is entirely supposable that the intermittent discharges of the choreic movement is due, first, to the natural tendency of the cell to discharge rhythmically, nerve force; and secondly, to the failure of that inhibition which in the normal cord prevents rhythymic discharge of nerve force.

A remarkable fact in connection with rhythmic choreic tremors is, that they may go on continuously without sensible fatigue; whereas, if a simple to and fro movement be made by the effort of the will, only a few moments will be required for exhaustion. The theory of the reversion of the cell to its original condition of rhythmic discharge, explains this fact; for we know that those cells which rhythmically discharge force do not tire. A normal heart continues to beat, it may be, through a century, without rest and without fatigue.

In regard to this theory of rhythmic discharge, I have

no evidence to offer the Society this evening; indeed have not thought much over the matter. For putting it aside, I set myself to thinking whether or not it were possible, in any way, to throw light upon the condition of inhibition in the spinal cord of the choreic dog. My first series of experiments was directed to determining whether the motor cells of the spinal cord in the choreic dog are still capable of being inhibited. I have found that when division of the spinal cord is made low down, during etherization, the shock of the operation prevents a repetition of the movements for a considerable period after the recovery of consciousness. Thus, in one experiment, after division of the cord in the upper dorsal region, the movements did not reappear for over a quarter of an hour after complete recovery of consciouness. They first made their appearance in the hind legs, and afterwards in the front legs. The movements were finally much more violent in the hind legs than they had been before section; but all synchronism between the front and hind legs was completely set aside by the section.

In the second experiment the movements were rhythmical, and, as is often the case, were most marked in the opposing front and hind legs. They were, as in the first case, immediately stopped by ether. The cord was cut in the lower dorsal region, and the movements reoccurred in the front leg as soon as the effect of the anæsthetic had gone off, but did not come back in the hind legs for over an hour after the return of consciousness. It is plain to my mind that the shock of section of the cord has an effect upon these movements. In the first experiment, the front legs were nearest the seat of section, and recurrence of their movements was longest put off. In the second experiment, the section was practiced very low down, and the hind legs were inordinately influenced. In the choreic dog, as in the child, the movements can certainly be inhibited temporarily by the cerebrum.

Another experiment, of which I herewith submit the tracing,

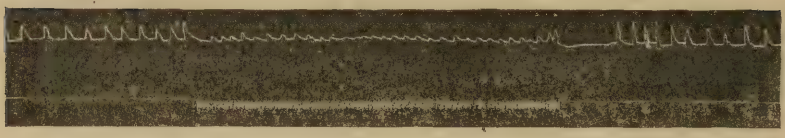

FIGURE 1. Widened space in lower line registered the application of faradic current to sciatic nerve.

confirms the fact that the function of inhibition in spinal chorea is still capable of being aroused. A complete section of the cord was made in a choreic dog, and although no evidence of pain was caused by very powerful faradization of the exposed sciatic nerve, the choreic movements were very markedly inhibited, as is shown in the photographic reproduction of the tracing made. The upper line represents the writing of the movements of the right leg, the lower line the time of application of the current. This experiment affords direct proof that there are inhibitory centres in the cord, or else that the ganglionic motor cells of the cord are directly inhibited by peripheral impulses.

In 1869, Dr. T. A. Chaperon proved that, in small doses, quinine caused in the frog a lessening in the reflex activity, which is relieved by high section of the spinal cord. The only explanation of this fact, which has been abundantly confirmed, is that the section has removed from the motor centres of the cord the influence of the stimulated inhibitory centres high up, and that therefore quinine is a stimulant to the inhibitory function of the cord. It is true that Dr. Sedgwick has combatted this theory, basing his opposition chiefly upon the fact which he discovered that atropine prevents the primary inhibition of reflexes by quinine.

It has, however, never seemed to me that the views of Sedgwick are entitled to much consideration; and the fact which he discovered is in accord with, and confirmatory of the theory of spinal inhibition. As first shown by Fraser, during recovery of the frog from atropine poisoning, there is a stage in which motor paralysis and

a reflex activity so excessive as to produce tetanus, coexist. The explanation of this as given by Fraser, has been proven to be incorrect by the experiments of Ringer and Murrell, whose views are summarized in the following passage taken from my Treatise on Therepeutics: "Drs. Ringer and Murrell believe that both the paralysis and the tetanus are due to a depressant action. The theory is that the normal cord has a power of resisting impulses received from the periphery, and especially of preventing their wide propagation among the spinal centres. During the first stage of the atropine poisoning it is supposed that the motor portions of the cord are so paralyzed as to be unable to form or propagate any motor impulse, and hence the general paralysis. Later on, however, the motor cells have so recovered themselves as to be able to generate impulses freely, although the resistive power of the cord is still in abeyance. Consequently a peripheral impulse plays as it were at will up and down the spinal cord, and instead of giving rise to a simple reflex action, gives origin to a series of reflex movements, involving all the muscles and constituting a tetanic convulsion."

It would seem, therefore, that atropine is a paralyzant; quinine, a stimulant of the inhibitory spinal function; and that one should antagonize the other is naturally to be expected.

The discovery of Sedgwick, that atropine brings back the reflexes arrested by quinine, is in verity a strong corroboration of the theory that the quinine acts upon the inhibitory apparatus. Holding, as I long have, that quinine is a stimulant and atropine a depressant of spinal inhibition, it occurred to me that if the choreic movements be due to weakness of the inhibitory centres of the cord, quinine should check the movements by strengthening the centres, whilst atropine should increase the movements by still further defining the centres.

I have made a number of experiments upon the action of quinine on the choreic movements in the dog, with results so concordant that it is certain that quinine, even

in small doses, does arrest the choreic movement. As evidence of this I offer to the Soceity the following tracing, which was made upon a revolving drum through a mechanical apparatus registering the movements of the paw of a choreic dog.

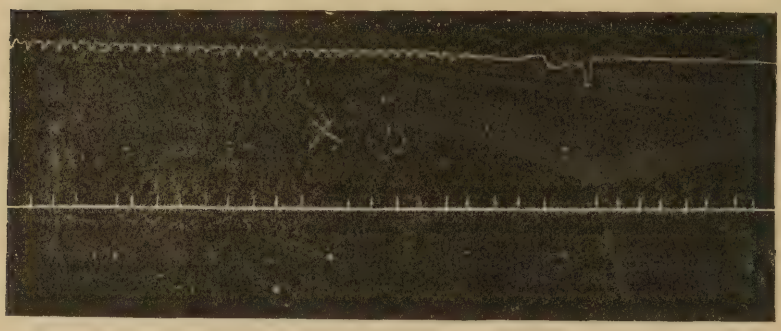

FIGURE 2. Lower line, seconds register. Upper line, register of choreic movement. Quinine injected at x.

With atropine I have had opportunity to make but a single experiment, not being able as yet to obtain another dog with chorea sufficiently pronounced to register itself well upon the drum. A tracing of this experiment I submit to you, and you will see that the results have been

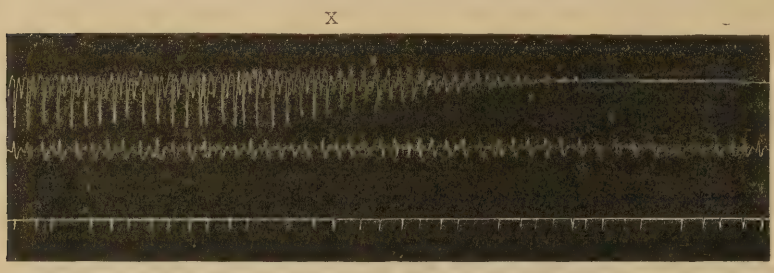

FIGURE 3. Lower line, second marker. Middle line, the register of choreic movement in the unpoisoned dog. The upper line shows same movements some minutes later after injection of atropine at position of cross. Quinine was intravenant injector.

remarkably decisive. The atropine quadrupled the extent of the choreic movements. You will notice, also, on

the tracing the registration of an immediate arrest of the movements produced by the injection of a small dose of quinine.[2]

Although in the choreic cord excessive movements occur as the result of the failure of inhibition, the partial, and finally it may be almost complete, paralysis which accompanies chorea shows that the motor cells themselves are functionally depressed, and indeed in my paper of 1885 it was shown that finally the motor cells, at least in canine chorea, undergo atrophic changes.

It is seemingly a priori, inevitable, that in chorea of a severe type the general muscular tone should be diminished, and that the reflexes, sharing in the general relaxation, should be less active, and in very severe cases even be abolished. The best explanation that has been offered of the phenomena of reinforcement of the knee jerks by voluntary movement, is that of the overflow; this widely accepted theory teaches that an influence destined primarily for a certain region of the cord has a tendency to spread itself throughout the cord. In the normal cord this overflow is more or less held in check by the inhibitory function of the spinal cord, and meets with resistance from the inhibitory function. A depression of the inhibitory function of the cord would therefore tend to diminish the resistance to the overflow, or in other words to increase the effects of the overflow. If the theory held in this paper in regard to the condition of the cord in the choreic movements be correct, we would expect to find in chorea, in the child, the knee-jerks lessened but the influence of reinforcement increased.

Knowing that my friend, Dr. Wharton Sinkler, had been engaged for some time in studying the knee-jerks in Sydenham's chorea, and knowing that his observations would be free from any possible influence by preconception, he having no knowledge of the inhibitory theory, herein propounded, I wrote asking him simply as to the

[2] Whilst this paper was going through the press a choreic dog having been secured it was found that atropine enormously stimulates the choreic movements.

condition of the knee-jerk in chorea, and received the following reply:

"I have examined the knee-jerks in the cases of chorea presenting themselves at the Infirmary for Nervous Diseases for several years. In the majority of cases the knee-jerk is diminished and in a few it is entirely absent except by reinforcement. Reinforcement in those cases where the knee-jerk is absent seems to produce excessive results, very strikingly shown in a patient at my clinic recently. This condition, I confess, I do not fully understand. One would rather expect exaggerated knee-jerks in chorea, as a result of the continuous muscular movement; supposing that these involuntary movements would have the effect of reinforcement. From this, one would infer that it requires a volitional act to cause reinforcement."

It is scarcely necessary to point out how these observations dovetail into the theory brought forward in this paper. As Dr. Sinkler says, the explanation of the reflex phenomena in chorea is extremely difficult with the older ideas of the origin of the choreic movement; that it is due to a simple excitement, of the motor cells. If, however, the choreic movement be of spinal origin, and if it be due really to a condition of depression of the spinal apparatus, no reinforcement is to be looked for from the choreic movement. It will be a matter of great interest to determine whether the choreic movement, so-called, which originate in the cerebrum, do or do not reinforce the knee-jerks. It seems to me a priori, therefore, that in cerebral chorea it will be found that the reflexes are reinforced by the motion.

As connected with the subject which I have been discussing, although I am not at present able to say what the exact relation is, I may call the attention of the Society to a discovery of Professor Reichert, of the University; namely, that it is possible to produce in the dog an artificial chorea. My friend, Prof. Chas. Richet, of the Ecole de Medecine, Paris, some years ago pointed out that if active artificial respiration be maintained, enormous

doses of strychnia injected into the veins of the mammal produce a complete paralysis. In studying this matter, Prof. Reichert has found that it is possible, by the use of the proper dose, to produce a condition in which neither the motor nor sensory nerves are able to respond to artificial stimulation, but in which the animal is violently choreic. The circulation is actively maintained but the reflexes are absolutely gone; so that violent irritation of the cornea elicits no response, and the most powerful faradization of the sciatic nerve produces no effect upon the circulation or upon the tributary muscles. Although sensory and nerve trunks are absolutely paralyzed, according to all ordinary tests, yet the motor nerve trunks still carry impulse from the centre when chorea occurs. I have found that quinine arrests these movements as it does the movements in the natural chorea of the dog. Atropine has not, in the various experiments which I have made, produced any distinct effect.

In concluding my remarks upon this subject, I want to distinctly say that the present communication has been made at this time partly on account of the interest of the subject, partly because I may have to wait months before I can get hold of another choreic dog, and partly because of the persistency of the secretary of the Society in his request for me to bring the matter before you. I do not hold myself in any way personally responsible for the correctness of the theory, and if further investigation shall disprove it, I will throw it aside as an old worn-out shoe.

The discovery of the power of quinine over the choreic movements has naturally led me to a trial of quinine as a remedy in chorea. I had, however, been only a few days since the present research was commenced. In the Clinic of the University Hospital, one case of marked chorea in a child presented itself. The child was cinchonized, and in two days the choreic movements had nearly stopped. As the case has not returned since it is probable that the relief continues.

Choreic dogs are frequently brought for treatment to

the Hospital of the Veterinary Department of the University of Pennsylvania, and speaking to Professor Pearson, he told me that at that time they had a very fine Irish setter suffering from chorea; that they had had a number of cases and so far failed to benefit any of them, and that he would try the quinine. Four grains of quinine have been given to the dog every three hours, at the present writing, for one week. The result has been almost complete disappearance of the choreic symptoms and extraordinary gain in power in the spinal cord. In the beginning of the administration the dog could scarcely stand; to-day he can walk some hundred of yards without difficulty. The future must, of course, decide whether these results are merely delusive coincidences, or whether quinine has curative power over chorea. Certainly, however, the quinine is worthy of trial.

HYSTERICAL SEIZURES RELIEVED BY HYPNOTIC SUGGESTION.

By JUDSON DALAND, M. D.,

Instructor in Clinical Medicine and Lecturer on Physical Diagnosis and Symptomatology in the University of Pennsylvania, Assistant Physician to the University Hospital; Physician to the Philadelphia Hospital and to the Rush Hospital for Consumptives, Philadelphia.

THE case that I wish to report this evening occurred in an unmarried woman, aged eighteen, living in the city. She says that her mother is living and well, and that her father died by accident. She has no brothers nor sisters. As a child she suffered each winter from tonsilitis. At the age of thirteen she had pneumonia, and later an attack of measles. At the time of teething there were no convulsions. She was always excitable, and invariably fainted at the sight of blood. Menstruation began at the early age of 10½ years; at first was irregular, sometimes occurring but once in two months, and at other times every *three* weeks. The pain was so great during the first few days of menstruation that she has been compelled to go to bed. The quantity of blood lost was considerable, and menstruation usually lasted ten or twelve days. She states that since about six months ago she has frequently suffered from belching after eating; occasionally there is vomiting, and often she is attacked with convulsive movements and unconsciousness immediately after taking food. The matters vomited were acid and bitter to the taste. Occasionally before and after menstruation the breath is fetid. She averages two movements of the bowels weekly.

On March 29th, 1892, at 7 p. m., the first attack occurred, following a vomiting spell. She fell from the chair to the floor entirely unconscious; the face was in convulsive movement, and the entire attack lasted ten minutes. She had no recollection of the attack, but felt

as if she had fainted. During the day there was nausea, and the appetite was impaired.

The second attack occurred six days afterwards, and in the fall she bruised her arm severely. This attack was like the first, but lasted one hour.

Four days later a third attack occurred, which was similar to the first. They have occurred as often as seven times in one day, and average one daily; she has noticed that fright is able to produce an attack.

Her attending physician, Dr. W. E. Robertson, witnessed one of these seizures, and he gives me the following description:

He found her in opisthotonos. This was followed by convulsive movements, especially the diaphram, accompanied by loud borborygmia. She remained unconscious twenty minutes. Anæsthesis was complete, and the introduction of pins under the nails produced no evidence of pain. The expression of the face was normal; the pupils were of average size, and seemed to vary with respiration; from time to time there were strong efforts to vomit. In case of these attacks she does not bite the tongue nor injure herself by falling, and on one occasion she described a vision that occurred to her.

She was hypnotized on September 6th by holding the laryngeal mirror nine inches from the eyes on a horizontal level, and by the aid of which light reflected from a student's lamp. Somnambulism was produced in four minutes. The eyelids dropped in the ordinary manner in three minutes, and complete hypnosis was secured in four minutes. Immediately prior to this the pupils rapidly dilated and contracted. At first the sleep was slight, but upon suggestion it became profound. Upon raising the right arm, after momentary hesitation, it dropped completely relaxed, as did the right. The suggestion was then made that the left arm was paralyzed, and that the skin on the dorsum of the left hand was anæsthetic. Puncturing the skin produced no objective sign of pain, and the patient said that no pain was experienced; whereas, when the corresponding cuta-

neous surface of the right hand was punctured the hand was immediately withdrawn, and pain was complained of. She was then commanded to raise the left arm, which was impossible; when commanded to raise the right arm this act was performed immediately. She was then told that, upon awakening, she would again become somnambulistic upon gazing into the eyes of the operator, which was accomplished. Finally it was suggested that no attack occur until the following Friday at 9 a. m., when she was again to be hypnotized. The following Friday she was hypnotized in the same manner in three minutes, and the same phenomena was reproduced. Again the suggestion was made that no attacks should occur until the following Friday. She came as had been agreed upon, but I was called away unexpectedly, and was unable to communicate with her. She reports that, upon entering the office, she felt a peculiar indescribable sensation like that experienced when in hypnotism. She returned the following Friday, but I was still at the Swinburne Island Hospital, and she reports that she experienced the same sensation.

On October 1st, or fifteen days after the last hypnosis, she had a seizure, excited by a quarrel with one of her relatives. On the succeeding day she had two attacks, which were excited by a great disappointment. This statement was made on October 6th, and when hypnotism began it was obvious that she was endeavoring to resist, but she became somnambulistic in less than four minutes. Her pulse was 132 per minute. No suggestions were made, but when sensation was tested it was found normal, with the exception of the skin on the left hand, which was completely anæsthetic. The left arm was in a condition of paralysis, but the other muscles responded readily to the will.

It should be remembered that this paralysis of motion and sensation in the left hand and arm was suggested at the first hypnosis, and they occurred voluntarily at this meeting. It was then suggested that the following morning at ten o'clock she should write a letter describ-

ing her condition, which she did, and a second suggestion was made that, in five minutes after awakening, she would complain of intense pain in the left ear, which would last but one minute. This was also successful.

On October 14th she was again hypnotized with the same result. After each hypnosis she expressed herself as feeling stronger and better. Two days before, she was hypnotized by Dr. Robertson, and remained in that condition twenty minutes. Each suggestion was obeyed, and she presented the same phenomena regarding the left hand and arm as has been previously narrated. This same member was rendered cataleptic by suggestion, and she was informed that two minutes after she awoke she would not be able to see anything for thirty seconds. At the expiration of two minutes she complained that she could not see, and attributed it to the glasses that she wore.

There has been no hysterical seizure since October 1st, and she considers herself in good health.

To-day, January 23d, she called and stated that she had had two very slight attacks from which she fell to the ground, but was perfectly conscious, and she also says that the attacks were totally different from the original attacks. They were superinduced in each case by severe gastric irritation. She was hypnotized in less than two minutes, and presented the same phenomena, and also obeyed a post-hypnotic suggestion.

Stated Meeting, January 23, 1893.

The President, Dr. FRANCIS X. DERCUM, in the chair.

Dr. H. F. HANSELL, read a paper entitled

THE POSSIBILITY OF THE EARLY DIAGNOSIS OF LOCOMOTOR ATAXIA FROM THE EYE-SYMPTOMS.

The eye symptoms of the earliest stage of tabes may be described under three heads, those arising from affections of the extrinsic muscles, of the pupil and of the optic nerve. A number of observations from clinical cases and pathological studies has been recorded to prove that partial or complete paralysis of any one of the straight or oblique muscles or the elevators of the lids of one or both eyes, is at times a fore runner of ataxia. The muscles most frequently affected are the external rectus and the levator palpebrarum giving rise to ptosis and crossed diplopia. A strong diagnostic feature between paralysis from tabes and that from other causes, is that in the former the paralysis is incomplete and transitory. In the late stages when the nuclei in the floor of the fourth ventricle become implicated in the sclerotic process the paralysis recurs and the condition known as ophthalmoplegia results. The cause of the transient paralyses is ascribed by a few writers to nuclear disease, but the majority incline to the view of a vaso-motor disturbance.

The pupils are studied with reference to their size and shape and their reactions. Interference with the conduction of the sense of light, with its preception by the vasal ganglia, with the nuclei in the floor of the fourth ventricle or their connecting fibres, with the cilia-spinal centre in the cord, or with the pupillary branch of the third nerve, or vaso-motor fibres of the sympathetic, will determine anomalies of the pupil. Myosis is a characteristic of the tabetic pupil and must be regarded as a vaso-motor paralysis originating in disease of the centre. Berger in an analysis of 109 cases of tabes, followed

through the three stages, declared that in all, the pupils were altered in size, and that this alteration was found in the great majority of cases, in the preataxic stage. The same writer and many others, have shown that the Argylle-Robertson pupil, not only is common in tabes, but is one of its earliest signs. A peculiarity of the contracted pupil of tabes is its medium dilatation to myonatics and the comparatively long time necessary for its return to its previous size.

Both myosis and the Argylle-Robertson pupil may precede altered locomotion and sensation by many years. The pupils may, also, be irregular in outline and unequally contracted.

The accomodation is seldom affected in the early stage. Two of the three cases briefly mentioned below have myosis and Argylle-Robertson pupils with no other spinal symptoms and showed other signs of tabes develop. They will furnish strong evidence in favor of the possibility of diagnosing that disease in its earliest stage, from the eye symptoms. The third case died from tabes eight years after myosis was first noticed.

The first sign of altered function of the optic nerve is limitation of the color field without narrowing of the field for white, deterioration of central acuteness of vision for form or opthalmoscopic changes. Later the field for white becomes concentrically limited, central vision declines, and the opthalmoscope shows the well known characteristics of non-inflammatory, incipient or advanced, atrophy. Emphasis should be laid on the superiority of the perimeter over the opthalmoscope in the early diagnosis of tabes. Dilatation of a retinal vein, which later returns to its normal calibre has been observed before evident involvement of the optic nerve. There is no doubt that incipient or even pronounced atrophy of the optic nerves may be a forerunner of tabes.[1]

Case I. Mr. B., age 61.—Neurotic history. Pupils very small and have been so for ten years. L. slightly irregular in outline. Both respond to convergence but not to light. Four months ago he commenced to see double from a paresis of the L. external rectus. The double images were fused at my first examination by a prism of 12°, later by 30°, and, at my last examination, made two weeks ago, by 4°. The lower, nasal retinal

[1] The presence of inflammatory exudation from the iris must be excluded and the repeated observations necessary to estimation of size be conducted with the same degree of illumination.)

vein was dilated, but soon regained its normal calibre. The color fields were temporarily limited, optic discs normal in color and outline. No general symptoms.

CASE II. Mrs. V., age 55. Unilaterial myosis and Argyll-Robertson pupil. Pain in lower extremities, no alteration of fields, no paralysis or incoordination.

CASE III. Mrs. G., age 60. Myosis, loss of color perception and night blindness preceding for eighteen months, spinal optic nerve atrophy. Death eight years after first examination, from tabes.

Conclusions:

1. Transient ptosis or external ocular paralysis may, be one of the earliest symptoms of locomotor ataxia.

2. The Argyll-Robertson pupil, inocular or binocular, may precede for a variable length of time the general symptoms of locomotor ataxia.

3. Idiopathic, non-inflammatory optic nerve atrophy may be the first manifestation of locomotor ataxia.

4. Transient ptosis or diplopia, Argyll-Robertson pupil, incipient optic nerve atrophy associated in an individual past middle life, of inherited neurotic tendency, are strong presumptive evidence of the first stage of locomotor ataxia.

DISCUSSION.

Dr. H. C. WOOD.—I should like to call attention to one point which I think has not been dwelt upon and that is that the Argyll-Robertson pupil is not rarely seen reversed; in other words, there is preservation of the ocular reflex for light and loss of the reflex for accommodation. I think that this is not so common as the true Argyll-Robertson pupil, but it frequently occurs and has not been much spoken of. It is probably as significant as is the Argyll-Robertson pupil of organic disease of the nervous system.

Dr. JAMES HENDRIE LLOYD.—Cycloplegia occurs in some forms of multiple neuritis which simulate tabes. It is also observed in post-diphtheritic paralysis. I do not think it is common, as Dr. Wood states, to have the reversal of the Argyll-Robertson phenomena in true tabes. I have, however, seen cycloplegia come on in one or two cases of multiple neuritis, post-diphtheritic, simulating tabes in some of the symptoms. This might be called, on hasty observation, a reversal of the Argyll-Robertson pupil, although of course it is not truly so.

I was struck by a reference in Dr. Hansell's interesting paper to a form of ophthalmoplegia, I think external, coming on with symptoms of migraine, in the early stage of locomotor ataxia. Charcot, in a clinical lecture, has reported a case of that rare form of disease, "migraine ophthalmoplegique." It occurs to me that there might be some connection between this migraine and late coming tabes, especially as it is claimed by some that migraine is not an altogether uncommon predecessor of tabes or even of general paresis.

Dr. WHARTON SINKLER.—My experience has been the same as that of Dr. Lloyd as regards the reversal of the phenomena of the Argyll-Robertson pupil. I have seen two or three instances of it in tabes, but I think that it is not often met with.

Dr. Hansell's paper has interested me greatly. I really think that when we meet with cases of transient diplopia or ptosis, we ought to suspect the on-coming of tabes. I saw only a couple of days ago a patient who is in an instance of the truth of what Dr. Hansell has said. The gentleman who even now has only incipient tabes, has myosis, absence of knee-jerk and slight incoordination, but he has no ataxic pains. He has, however, well-marked disturbances of sensation with loss of thermal sense. On inquiry, he told me that several years ago, he had attacks of diplopia which were attributed to indigestion. This was six or seven years ago, before the other symptoms of tabes were observed.

Dr. J. MADISON TAYLOR.—Some time ago, I showed to the Society, a boy with Freidreich's ataxia, in whose case, the pupillary symptoms were reversed. I do not know whether or not this has any significance in that form of ataxia. In looking over the literature, I find the pupillary symptoms of Freidreich's ataxia are rather irregularly stated. In this case they were absolutely reversed.

Dr. CHARLES K. MILLS.—Dr. Hansell has given an excellent resume of these pre-ataxic eye symptoms, and the only remark that I have to make is that perhaps too much stress might be laid on these ocular symptoms in making the diagnosis of future ataxia. This might be misleading. It is true that these symptoms do frequently precede this disease, but on the other hand, it is true that in a fair percentage of cases, one or more or all of the ocular symptoms which have been mentioned, may be the precursor of some other form of degenerative disease,

or indeed so far as the symptomatology is concerned may be the only clinical picture of the degenerative disease itself. While of course it is valuable to emphasize the importance of these symptoms, a word of caution should I think be given with reference to hastily making the diagnosis of coming ataxia from their presence. Myosis is seen in paretic dementia. It also occurs in pseudo-paretic dementia, of syphilitic origin, and also in some forms of multiple neuritis.

Dr. H. C. WOOD.—I did not mean to be understood as saying that the reversed Argyll-Robertson pupil, but only that it was not very rare.

I recently reported in a clinical lecture to be published in *La Semains Medical* a case in which the diagnosis rested partly upon the pupillary condition. The case, I believe, is one of a new form of locomotor ataxia crisis which I have proposed the name of ataxic lympho-pathy. The patient is a man with a long history of exposure to the elements as a boat captain. When 58 years of age, he was seized with a frightful pain in the left angle of the jaw. This was associated with great swelling. He consulted his family physician, who believed that there was going to be suppuration of the gland. The pain amounted to agony. In three or four days, however, the swelling subsided without leaving any trace. Shortly afterwards the man had a similar attack and this has been followed by repeated attacks with frightful pain, with swelling of the glands, this swelling subsiding in a few days and the gland apparently going back to its normal condition. as a result of a series of the attacks, however, the cervical glands have become slightly enlarged.

Without going into the details of the case, I found that the character of the pain and swelling indicated a neurotic origin and this led to the suggestion that possibly I had a form of ataxic crisis to deal with. I found myosis with reversed Argyll-Robertson pupil, complete loss of the knee-jerks and marked retardation of the sensation in the legs with pronounced parasthesia, and that during the last three years, the man had gradually lost the power of equilibriation. I do not see how this case can be looked upon as anything else but a case of locomotor ataxia with crises affecting the region of the lymphatic glands, with enlargment of these glands.

The PRESIDENT.—I should like to remark that the history of swelling and painful crises related by Dr. Wood is somewhat like that of the cases of Adiposis

Dolorosa, which I have reported, in which was swelling of the tissues and in which shooting and darting pains accompanied the pain. Afterward the swelling would somewhat, though not altogether, subside. It is possible that Dr. Wood's case might be explained on the theory of neuritis.

Dr. H. C. WOOD.—In the case to which I have referred, there was no tenderness over the nerve trunks and there was evident enlargment of the lymphatic glands. Neuritis would hardly explain the loss in the knee-jerks, the retardation of sensation and the condition of the pupils.

Dr. H. F. HANSELL.—With the reversion of the Argyll-Robertson phenomenon, I should look for a different pathology. The paralysis of the sympathetic fibres to the pupil from destruction of or pressure upon the cilio-spinal centre, is accepted I believe, as the cause of the Argyll-Robertson pupil. If the condition is reversed, we should have to explain it by disease of the nucleus of the iris in the floor of the fourth ventricle.

Dr. Lloyd's statement that paralysis of the accomodation is found in central diseases of the nervous system is true, but that fact does not bear upon the eye symptoms found in the earliest stages of the ataxia since the accomodation is seldom affected in that disease. The pupil, which has its sympathetic centre in the higher part of the spinal cord, is the only one of the muscles within the eye, commonly involved.

In conclusion, I wish simply to say that my paper was designated "the possibility," and that its conclusions are not absolute because a large material is hard to collect and the cases difficult to follow to their termination, for reasons that will readily occur to you. The association of the three symptoms, however, transient paralysis of the external muscles, Argyll-Robertson pupil and loss of green in the visual field, would warrant a careful study, and search for other symptoms of tabes.

Stated meeting, February 27, 1893.

President, Dr. CHARLES K. MILLS, in the chair.

Dr. CHARLES L. DANA, of New York, read a paper entitled:

A CLINICAL AND PATHOLOGICAL STUDY OF SHAKING PALSY, WITH A REPORT OF TWO AUTOPSIES.

In the clinical part of his paper, Dr. Dana gave the results of examinations of the blood, using Ehrlich's stains; these showed the presence of a progressive chlorosis, but not an anæmia; also some changes in the myelocytes. A number of sphygmographic tracings of the pulse were shown, and the conclusion reached, that there is in paralysis agitans, a general vaso-motor paralysis affecting the skin, viscera, nerves, and lower nerve-centres, but not the brain.

In the pathological part the author first went over the literature of the pathological anatomy of paralysis agitans giving a summary of the findings in all authentic cases. The number of available cases was found to be only fourteen. The findings in late years were much more uniform and definite. The author then reported his own two cases, and gave the results of his microscopical examinations. These, in brief, revealed the presence of great vascular dilatation, connective tissue, proliferation, and nerve-cell degeneration. The lesions were most marked in the central gray of the cord, and the vagus, and glossopharyngeal nuclei. The brain cortex, internal capsule, and basal ganglia were normal.

DR. Dana believed the vascular and connective tissue changes to be primary, the nerve-cell changes to be secondary. He thought the process resembled a low grade of chronic inflammation. At any rate, he considered the primary cause of paralysis agitans to be a toxine of autochthonous or microbic origin. Shaking palsy was a toxine disease. The primary seat of the trouble was in the spinal cord, more particularly in the central

and anterior gray matter, supplied by the central arteries.

Dr. Dana thought that the time was past when paralysis-agitans could be called a functional disease without a morbid anatomy. The most recent and thorough post-mortem studies showed results in conformity with those reported by the writer. The practical conclusions in regard to therapeutics, were that remedies for the disease in in its earlier stages should and probably would be found. Such remedies would be of the character of anti-toxine or diathetic drugs modifying metabolism and promoting excretion. Some results of the author's therapeutical experience were given.

DISCUSSION.

Dr. FRANCIS X. DERCUM.—I infer from what Dr. Dana has said that he places on the changes which he finds in paralysis agitans rather the significance of terminal than the significance of causal changes. One of the points about his paper that strikes me as subversive of our former ideas is that he describes the disease as of spinal origin, and that we have a tremor of spinal and not of cortical origin. Tremor has always been looked upon as indicative of cortical and not of spinal disease.

We are still absolutely in the dark as regards the primary cause of this so-called functional trouble. Dr. Dana believes the cause to be a toxine, but this is evidently speculative only. It is to my mind fitting that in a disease in which we have motor symptoms, that we should have terminal changes in the motor tract. We have, as Osler pointed out in a paper read before this Society, terminal changes in the motor tract in other diseases such as the hopeless chorea of adults. It may be that there is some poison circulating in the blood, which leads to these peculiar changes, but there is as yet no evidence of it.

Dr. JAMES HENDRIE LLOYD.—It seems to me that the vital suggestion in Dr. Dana's paper is that of a microbic or humoral pathology of this disease. We recall that first hydrophobia, then tetanus and more recently chorea have come within the domain of the modern microbean pathology. It remained for Dr. Dana to come to Philadelphia and give us this original suggestion of some vito-chemical change in the blood; probably of microbic origin, being the cause of shaking palsy.

I cannot help thinking that some of the changes which Dr. Dana has demonstrated are rather commonplace, and possibly are identical with what we might find in the cords of many persons of the age of his patient. They certainly are not suggestive of any very distinctive disease process. Slight thickening of the blood vessels, increase in the neuroglia, and increase of the epithelium of the central canal, are rather indefinite conditions on which to establish a distinct disease process. They are terminal changes which might be attributed to a variety of irritants in the blood. The one vital suggestion is as to the possibility of this being of vito-chemical or microbic origin. The proof of the correctness of this hypothesis must be sought in systematic experimentation in the biological or bacteriological laboratory. At present it is simply an ingenious hypothesis resting upon not very broad anatomical grounds.

Dr. JAMES TYSON.—In considering the toxic origin of paralysis agitans, one is met with the thought that in diseases of acknowledged toxic origin, we have ordinarily a shorter duration than in this class of cases, with the exception of lead poisoning; while it might be expected, also, in such a disease as paralysis agitans, that the large number of cases observed would have permitted one to have established some such causal relation, if it were present.

Dr G. BETTON MASSEY.—After hearing this interesting account of the pathological changes, I feel that, personally, I shall approach the treatment of paralysis agitans with more hope of effecting results. I was one of those who believed that there was no pathology recorded in literature, and, from what I have observed, the treatment of others seems to be based on the same view, being directed toward a peripheral effect, whether internal or external remedies are used, or electricity employed. Attempts are often made to lessen the irritability of the centres by the use of Franklinic sparks, and I have seen pain apparently lessened by that form of treatment, but no arrest of the progress of the disease.

This new suggestion in regard to the microbic origin of the disease, while very interesting, does not help us a great deal, since we are already face to face with the fact that but few microbic diseases are capable of being treated directly, in spite of a positive knowledge of their microbic origin.

It appears to me that we must direct our treatment

towards the removal of the result of microbic action by stimulating nutritive changes. If further attempts are to be made in the electrical treatment of this disease, we should direct our work to the passage of strong currents through the spine. I am continuously increasing the strength of currents used to the nerve centres of the spine, and I feel sure that therein we have a large field of usefulness. We have also, in these interpolar electrical applications, a means of carrying drugs into the body, by the recently revived process of cataphoresis, by which a more profound impression may be made on internal morbid processes.

Dr. J. MADISON TAYLOR.—I would ask Dr. Dana, if, in his study of this disease, there have been revealed to him any points bearing upon the early recognition of this affection and its differentiation from other forms of tremor. If there is any hope of doing good in such a disease as this, it will be in the early stages before degenerative changes have taken place.

Dr. A. A. ESHNER.—I could wish that Dr. Dana had used the term intoxicant rather than toxine in speaking of the etiology of this affection. The development of a toxine seems to commit one to the view of a microbic origin. Probably a better word still, in the present state of knowledge, would be "irritant." It seems to me that the disease belongs to the degenerative period of life, in which changes analogous to those described occur in many organs. One cannot, therefore, be surprised to find in the cord changes similar to those which we find in spleen, kidney, liver, and blood-vessels. I should have been glad if the lesions found had conformed with those of a diffuse sclerosis, affecting not only the cord but the cerebrum as well, and not necessarily confined to any group of cells or system of fibres. In conformity with this line of thought, a vascular irritant would have suggested itself in an etiologic connection. Still another point. Dr. Dana stated that paralysis agitans presents no cerebral symptoms, but it seems to me that there are occasionally cerebral symptoms of which impairment of memory is, perhaps, the most prominent. The delayed cerebral psychic tremor, sometimes noticeable, may, perhaps, be explained by an interference with the transmission of psychic impulses, as a result of changes in the conducting fibres analogous to those that are conceived to be responsible for the muscular tremor by interfering with the transmission of motor impulses. It seems to

me that by the assumption of an intoxicant or irritant, dependent upon defective elimination by the emunctories, we need not extend the range of speculation to such a limit as to have to include a possible microbic origin.

The PRESIDENT.—It seems to me that in some respects the discussion is hardly just to the paper. Some of the suggestions are new. We cannot say that only terminal or the ordinary degenerative changes have been described. The histological changes of degenerative disease will almost necessarily be the same or similar, however they may be described. Dr. Dana has suggested to us something definite. The extent and the location of the most intense changes which he found are different from those found in other degenerative diseases. The suggestion as to place of initiation of the changes; the question of the degeneration of the lateral columns of the cord; the fact that he has shown that in this one case at least no definite changes were found either in the cortex or in the cerebral tracts, or anywhere above the pons, and also that he has pointed out that the changes were especially marked in the nuclei of certain of the cranial nerves, are of considerable importance, and some of them have a decided bearing upon the peculiarities of symptomatology.

Dr. C. L. DANA.—When gentlemen now-a-days speak scornfully of microbe and microbic products, I am reminded of the Rev. Mr. Jasper, and his assertion that "the sun do move." And I am content to allow those who enjoy thinking that microbes are still theoretical things to join the ranks with the Rev. Mr. Jasper. I believe thoroughly in their pathogenic importance myself, and there is nothing of late years that has advanced us so much as bacteriology, or that gives us so much hope for the future of therapeutics. At the same time, I did not say that paralysis agitans was a microbic disease. I said that it probably was a toxic affection, and that the toxine was either of microbic or of humoral origin; and by humoral origin, I mean the same kind of origin as is supposed to produce the changes in rheumatism and gout, which we take to be the result of some defect in tissue metabolism or glandular activity.

These changes have been spoken of as terminal changes. All anatomical changes are terminal. The changes which you find in the lungs in pneumonia are terminal. They do not cause pneumonia. There is something that causes the anatomical changes. No one

thinks that pneumonia is caused by the presence of leucocytes in the air cells. I fancy also that the changes in Bright's disease are terminal, due to the constant irritation of the parenchyma of the kidney.

It has been intimated that the changes which I have described as occurring in paralysis agitans are of the same nature as those found in senility. I have considered this point in the paper, but did not dwell upon it when reading. My own experience is that the changes are not the same. I have examined the cords of old persons, and have compared them with the cords from these cases. The arteries are not thickened here as in senility, and there are peculiar changes in the cells. I think that the changes are such as would be produced by an irritant acting upon special parts of the spinal cord.

I can only say with reference to tremor that, so far as my experience goes, the tremor produced by irritation of the cortex of the brain, is a fine tremor of vibratory character, and that, when a coarser tremor has been observed, it has been due to some lesion lower down. I have had cases of myelitis where there was distinct coarse tremor like that seen in paralysis agitans.

Adjourned.

TETANY, WITH REPORT OF A CASE.[1]

By J. W. McCONNELL, M.D.,

Assistant Physician to the Epileptic Hospital of Philadelphia; Instructor in Electro-Therapeutics in the Philadelphia Polyclinic.

THE comparative infrequency with which tetany occurs in this country, the interesting phenomena presented, the necessity of recognizing it, and its obscure pathology, are my reasons for reporting the following case:

H., female, aged thirty-three years, unmarried, tailoress, a native of Ireland, with a family history exceptionally good. She had no illness except those common to children. She commenced menstruating when twelve and and a half years old, has always been perfectly regular, and other functions are normally performed.

Her present illness occurred December 8, 1892. A few days before, she was caught in the rain and wet through, and as a result, seemed to be suffering from a severe cold, stoppage of the air passages, slight discharge from the nostrils, irritation of the mucuous membrane of the pharnyx, and slight swelling of the tonsils. The usual home remedies were used, and the trouble seemed to be relieved.

On the day of the first attack of spasm, December 8th, while at her work she commenced to feel ill with headache, vague pains throughout the body, and a general sensation of numbness and prickling. About four hours after the first feeling of illness she was seized with tonic contractions of the muscles, first of the right hand and arm, and afterwards gradually involving the left hand and arm, both lower limbs, tongue and diaphragm.

At first the fingers were flexed at the metacarpo-phalangeal joints and extended at the carpo-metacarpal and phalangeal joints, with the thumbs adducted and opposed to the index finger. Later the hands became flexed on the wrists, giving a cup-shaped appearance to the hand. The muscles of the forearms and arms were all involved, causing a rigidity of the upper limbs in a

perfectly straight position. The muscles of the shoulder seemed to be affected, also the right sterno-cleido-mastoid, the head being drawn somewhat to that side.

The lower extremities were not affected to so great a degree as the upper. The feet were in a condition of extreme extension with the toes flexed. The thighs and legs were extended and straight, all of the muscles of the lower limbs seeming to be in a spastic state of almost equal degree. The muscles of the abdominal walls were contracted, the recti standing out very prominently.

The only muscles of the face that showed marked involvement were the zygomatics, and in consequence there was a peculiar smiling or sardonic expression. The tongue became stiff, making articulation very difficult, and when attempted causing pain in the tongue.

A hypodermic injection of morphia (grain $\frac{1}{4}$) was given, but before any effect was noticed the diaphragm was attacked with spasms, causing difficulty of respiration, and quickening the pulse. In a few minutes all the symptoms were relieved, and the patient dropped off to sleep, having been in the condition quite an hour. Fifteen grains of bromide of sodium, combined with eight grains of chloral, were ordered to be given every two hours. Voluntary motion caused great increase of the spasms and pain. The muscles were intermittently relaxed and contracted. Pressure on the brachial artery and nerves caused intensification of the spasm in the forearm and hand. There was free perspiration but no increase of temperature as taken in the axilla. The second attack lasted about thirty minutes, and left the patient in a condition of extreme fatigue, with sensations of soreness in the muscles. She had no third attack, but on the next day she developed symptoms of throat trouble, and for a few days suffered from an attack of follicular tonsilitis. Up to the present time, nearly four months after the onset, there has been no return of any sign of the muscular condition above described, and the patient seems to be in perfect health.

The manner of onset, the absence of history of traumatism, the absence of cerebral disturbance, the fact of the trouble having commenced in the extremities, the retention of consciousness, the bilateral involvement, the intermittent character of the attacks, and the increase of the spasm when pressure was made on the artery and nerves, were the points by which the diagnosis was made.

There was no foundation for a supposition of ergotism, and organic brain disease was excluded.

Three points in this case of most interest are: the severity of the attacks, especially the first; the rapidity with which the patient recovered; and the subsequent throat trouble.

From the reports of cases, the spasms seldom involve the muscles of the abdomen, face, tongue and diaphragm. Trousseau [2] recognized three classes of the disease, separating these according to the severity of the symptoms. In the first he places mild cases affecting the limbs solely; in the second, those in which the muscles of the face and neck were involved; in the third, those in which the spasm was both very severe and widely diffused. This case properly belongs to the third class. Perspiration, which occurred in it, is not present in the other classes. Dance [3] speaks of one case in which the perspiration was so profuse as to form a cloud of steam about the patient. In many instances abundant perspiration marks the crisis of the attack, and in this case it became noticeable just previous to the relaxation of the spasm.

In most cases of tetany the condition of spasm will last with varying severity and periods of recurrence for weeks or even years. Erb [4] regards cases that terminate in a few days as a great rarity, and thinks that usually a long time elapses before complete recovery takes place. Althaus [5] says that most patients recover in two or three months, especially under favorable circumstances. Gowers [6] claims that a case of any degree of severity may last only a few days. The subject of this report had but the two attacks occurring within twenty-four hours, and I have seen her from time to time until three weeks ago, and there has been no recurrence in the slightest degree.

[2] Trousseau: Quoted by H. N. Lyman, *Trans. Assoc. Am. Phys.*, Vol. i. Reported in *Med. News*, 1886, Vol. ii., p. 13.

[3] Dance: Quoted by Althaus, *Med. News*, 1886, Vol. ii., p. 485.

[4] Erb: Ziemssen's Cyclopedia of the Practice of Medicine. Vol. xi., p. 375.

[5] Althaus: Tetany and Tetanilla, *Med. News*, 1886, Vol. ii., p. 470.

[6] Gowers: Manual of Diseases of the Nervous System, Am. Ed., Phila., 1888, p. 1054.

The history of an acute cold preceding the attack, and the subsequent quinsy, suggest that the disease may be of toxic or infectious origin. Shattuck[7] reports a case in which the symptoms of tetany were noticed immediately after recovery from diphtheria.

Stewart[8] gives an account of a patient who had been suffering with chronic diarrhœa for ten years. Symptoms of tetany made their appearance two years after the commencement of the intestinal trouble, and were associated with exacerbations of that condition. In this case, also, there was a possibility of a myxœdemoid state.

Kussmaul[9] describes the case of a boy six years old, whose surroundings and food were of the poorest kind, and in whom tetany occurred in connection with a bad diarrhœa. The first attacks were relieved, but three months later he had another spell of vomiting and purging and the tetany returned. Althaus describes a like cause in a child three years old, who had attacks recurring every few hours.

Ord,[10] of London, exhibited a woman with symptoms of myxodema and peculiar symmetrical contractures of the flexor muscles.

Removal of the thyroid gland appears to be an exciting cause of a very fatal form of tetany. But little is known of the function of this gland, but experiments have shown that removal of one half of it does not produce tetanic symptoms. When, however, the whole gland is extirpated, symptoms of tetany quickly appear and are often quickly followed by death.

Stewart,[11] who studied the condition in animals, offers the theory that the function of the thyroid gland may be to remove from the blood matter which, if retained, would be injurious to the nervous system.

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[7] Shattuck: *Boston Med. and Surg. Jour.*, 1889, p. 231.

[8] Stewart: *American Journal Medical Science*, 1889, Vol. xcviii., p. 549.

[9] Kussmaul: Quoted by Althaus, *Medical News*, 1886, Vol. ii., p. 486.

[10] Ord: Quoted by Althaus, *Medical News*, 1886, Vol. ii., p. 484.

[11] Stewart: *American Journal Medical Science*, 1889, Vol. xcviii., p. 553.

Gowers[12] speak of diarrhœa, either acute or chronic, as one of the most fertile causes of this disease. Lyman[13] contributes a case in which the tetany appeared in a man who was affected with phthisis. Trousseau[14] mentions a case where the patient eventually died of phthisis. Müller[15] reports cases in which dilatation of the stomach was also found. Other writers speak of the probability of sepsis, rickets, the puerperal state, fevers, and other conditions in which a toxic condition might be originated. Erb,[16] in speaking of the nature of the disease, regards "the increased electrical excitability seen in it as due to molecular changes in the nerve substance. These changes are probably caused by delicate trophic disturbances occasioning great increase in the excitability of the motor apparatus, hence spasmodic attacks should be expected to occur whenever any unusually strong stimulus affects the motor nerves. Such a stimulus may be found in the voluntary efforts to perform muscular movements."

Althaus,[17] who seems to have made a thorough study of tetany, tends to regard it as due "to an undue and exalted state of excitability of the gray cells in the anterior cornua of the cord, and the motor nuclei in the medulla oblongata in accordance with the localization of the symptoms."

The exact pathology of the disease is as yet unknown, but Hirt,[18] Dana,[19] Gowers,[20] and others, mention the possibility of the disease being peripheral in origin, the seat of the lesion being located in the gray matter of the spinal cord. Erb mentions the trophic disturbance, and as a suggestion as to a possible cause for these changes I offer the query: can the disease be due to the absorption of toxic products, the result of putrefaction or infection?

[12] Gowers: Op. cit. p. 1050.
[13] Lyman: *Medical News*, 1886, Vol. ii., p. 487.
[14] Trousseau: Quoted by Althaus, *Medical News*, 1886, Vol. ii., p. 487.
[15] Müller: *American Journal Medical Science*, 1889, Vol. xcviii., p. 552.
[16] Erb: Op. cit., p. 373.
[17] Althaus: *Medical News*, 1886, Vol. ii., p. 488.
[18] Hirt: The Diseases of the Nervous System, N. Y., 1893, p. 486.
[19] Dana: Text-book of Nervous Diseases, N. Y., 1892, p. 444.
[20] Gowers: Op. cit., p. 1056.

Stated Meeting, April 24, 1893.

The President, Dr. CHARLES K. MILLS, in the chair.

Dr. WM. J. TAYLOR, Presented

A CASE OF DEFORMITY OF LEFT FOOT IN A CHILD ELEVEN MONTHS OF AGE.

The family history is perfectly good in every respect and except the deformity of the left foot, for which she is brought here to-night, she is in perfect condition.

As is seen, the left foot is much enlarged, not only in length, but in bulk, especially the anterior portion.

The first, second and third toes are much enlarged, from five to six times their natural size. This is especially marked in the second toe, which is much longer and thicker than the first toe. This enlargement includes both the phalanges and soft parts and the metatarso, phalangeal articulation as well.

She came first some six weeks ago to the Orthopedic Dispensary of the University Hospital.

The mother says the deformity existed at birth, but has increased very much within the past few months, but no increase since first seen. There is no pain or discomfort whatever.

The doctor never had seen such a deformity before, and he brought her here to-night in the hope that some member of the society may aid him in his efforts to form a diagnosis.

DISCUSSION.

Dr. FRANCIS X. DERCUM.—I can speak of this case only in a negative way. We have all of us recently been interested in dystrophies and especially in acromegaly, and the question naturally suggests itself whether or not this is a case of that character, perhaps irregular in type. I think, however, we must dismiss the suspicion of acromegaly. We have here a local deformity and one that is limited to a subdivision of one extremity, while in acromegaly all the extremities are more or less enlarged, and the enlargement is uniform in all directions except in length.

No one has suggested an explanation of these peculiar deformities which are exceedingly rare. The only case that I recall at present is one reported by an Indian medical officer some years ago, in which a child had one toe as large as the adult finger while the other toes were of normal size. As regards the pathology of these cases we can only speculate. If there be such things as trophic centres, as there is every reason to believe, it may be that future investigations will show some deformity in the centres which govern the growth of the affected part.

Dr. J. MADISON TAYLOR.—In a more than usually large experience in diseases of children, I never saw a similar deformity but once. This was a patient of the late Dr. Hayes Agnew, and I made drawings from life at his request, which are still in my possession, and should have been exhibited to-night as the conditions are strikingly analogous. The left foot, as here, was involved. The second and third toes had united in a solid mass to the very ends, making one huge toe, but with two nails, and projected nearly an inch beyond the normal limits. If my memory serves me this double toe had one jointless bone appearing like two bones cemented together, even as a double-barrelled gun.

In many points it closely resembled the case my brother exhibited to-night. Dr. Agnew removed these by amputation.

Dr. J. K. MITCHELL presented

A CASE OF LOCAL CATALEPSY. AN UNDESCRIBED NERVOUS DISORDER, PROBABLY HYSTERICAL IN NATURE.

The following very curious case I believe to be one of an entirely undescribed disorder, for which, in want of a sufficiently descriptive name, I have suggested the title of "local catalepsy," although this does not cover all the symptoms. The patient, a hearty girl of seventeen, a native of this state, was seen March 28th, and had every appearance of perfect health. Her left hand was two or three times, in December, 1891, and thereafter, suddenly weak, dropping the articles it held. A slight cut of a knife at the base of the left thumb in July, 1802, was followed by symptoms out of proportion to the injury, and suggesting the possibility of a local poisoning. The

whole hand swelled, ached and grew red and hot, and an abscess discharged in the region of the wound. After this the fingers gradually became stiff and assumed the condition described below, in which they have ever since remained. The fingers are about one-fourth flexed, the ulnar fingers slightly adducted, the fourth lying a little on the palmar surface of the third, the thumb in contact with the palmar surface of the index, a posture which, I shall call for brevity "the obstetric attitude."

The wrist has voluntary motion; the fingers none. Even when it shares, as sometimes happens, in the rigidity of the fingers, it still can be moved at will. The fingers may be passively placed in any position, and will retain it indefinitely. They are passively moved with great difficulty, against a constant waxy or rather lead-pipe-like resistance, which yields slowly and without jerk to the force used. Whatever posture of flexion, extension or abduction the fingers are moved into, it is with equal difficulty, and they remain in it until the patient voluntarily moves them with the right hand into the obstetric attitude, which is their most comfortable position. She thus moves them after thirty or forty minutes, because any other placing of them, if long continued—and especially if it be a position of extension—causes, after a time, pain in the wrist.

Sometimes the fingers pass slowly of themselves into an extreme abduction and extension, and so remain a long while, gradually returning again to the formerly-described attitude of easiest rest. This is the nearest approach they ever make to a voluntary movement.

There is analgesia of the whole hand below the annular ligament. There is no touch-sense or perception of temperature change in the hand. A pin-prick, unless deep, does not bleed. There is no evidence whatever of neuritis or degenerative neural change, nor any nerve tenderness, except a spot of tenderness over the median on the flexor aspect of the wrist, just above the annular ligament.

The nails have not grown so as to need cutting in three months past. They look like the nails of the right hand, which has no anomaly of sensation. Electric contractility is alike and perfect in both hands. The rigidity persists during sleep.

There were absolutely no evidences of hysteria in the patient's person, history, or manner, and the eye-grounds and color-fields were normal.

In spite of some want of direct evidence of hysteria it must be concluded that this is a hysterical case. In no other way can the involvement of *all* the nerves of the hand in a functional derangement of this kind be accounted for, with no degenerative or inflammatory changes, neural, spinal or cerebral, to be found. Nothing else could present such paralysis and so abruptly outlined a loss of sensibility, with no alteration of nutrition.

While to call it, as I have provisionally suggested, "local catalepsy" is only an attempt at naming it by its most striking feature, this does not describe inclusively all the symptoms presented, but as I can find no report of any case at all similar, this title will do until some better one is found.

DISCUSSION.

Dr. JAMES HENDRIE LLOYD.—It seems to me that the correct diagnosis of this case is hysteria. The association of segmental anæsthesia sharply defined, (a symptom which French observers insist upon as one of the most characteristic hysterical stigmata, especially when it exists in a paralyzed limb) with non-bleeding on pricking with a pin, suggests in the most direct way hysteria. An interesting thing would have been to etherize this young woman. I have now under my care a young woman with hysterical contractures of both limbs below the hip, but under ether they disappear. It is of first importance in all such cases to make careful search for all the hysterical stigmata. The element of heredity ought always to be inquired for.

Dr. FRANCIS X. DERCUM.—I would ask Dr. Mitchell why the patient returns the fingers to their ordinary postion after they have been in a constrained position for fifteen or twenty minutes? Did stroking of the muscles of the forearm, which must have played some part in the position of the hand, bring on a rigidity of the wrists?

Dr. JOHN K. MITCHELL.—I did not trouble you with a detailed report of the examination of the case, or give all the reasons that it led me to the conclusion of hysteria. I thought, however, that I had said that I could find no hysterical stigmata. The patient is a stout, blooming, ruddy-cheeked, well-nourished country girl. The family history is most excellent.

The reason that she changes the position of the

hand is on account of pain in the wrist. There is no pain in the hand. There is no paralysis in any proper sense of the word. I did not try the effect of stroking of the muscles.

Dr. J. W. McConnell read a paper on

TETANY, WITH REPORT OF A CASE (See page 79).

DISCUSSION.

Dr. Francis X. Dercum.—The third case presents so many difficulties that I could hardly attempt to solve it. I can not understand why this man with trigeminal palsy should have difficulty in swallowing, or why liquids should enter the trachea or regurgitate through the nose. If we look upon the case as of nuclear origin we are also involved in considerable difficulty because the trigeminal nucleus and the glosso-pharyngeal and the facial nuclei while relatively close together or not in such juxtaposition, that if trouble spread from the motor nucleus of the fifth to the glosso-pharyngeal, it would involve only a few cells in the nucleus of the facial. I cannot understand why there should not be complete facial paralysis at the same time.

The case of the negro is an interesting one, inasmuch as it involves the diagnosis of pressure at the base of the brain or nuclear palsy. To determine what is actually the matter in a given case is, I believe, something impossible. I recall a very interesting patient in which I at first thought that the lesion was nuclear but the rapid improvement which followed in twenty-four or forty-eight hours, the administration of the iodides showed that the case was specific and probably due to pressure at the base of the brain.

Dr. Amelia Gilmore presented

A CONTRIBUTION TO THE STUDY OF INSANITY AND NEPHRITIS (See page 90).

DISCUSSION.

Dr. Wharton Sinkler.—I would ask what proportion of these patients who were suffering from nephritis had an alcoholic history. So many of the patients

brought to the Philadelphia Hospital, both in the insane department and in the general wards, have an alcoholic history that I think this fact should be born in mind in considering the statistics of the classes under consideration. It is well-known fact in all departments of the Philadelphia Hospital that it is a rare thing to make an autopsy and find healthy kidneys.

Dr. FRANCIS X. DERCUM.—As I understood Dr. Gilmer, she regarded the kidney change as commitant rather than casual, and that is the position that many take. That we may have mental symptoms from the acute kidney affections there can be no doubt, but when we consider the immense number of cases of kidney disease outside of insane asylums and in which there are no mental symptoms, we must be cautious in considering the relation of insanity to renal disease. We must also remember that in the chronic insane all the organs are liable to show degenerative changes.

A clear history is not always obtainable, and some allowance must be made for the class of people, but that a majority of these cases are alcoholics is not true. The exact proportion with such history I can not give.

Dr. JAMES HENDRIE LLOYD exhibited a photograph of

A CASE OF HEMI-FACIAL ATROPHY.

The patient is a young Danish woman, aged thirty-three years, who has hemi-facial atrophy involving the scalp and extending down on the right side of the forehead to a region almost between the eyes, and extending as a scar-like tissue on to the cheek beneath the eye. There is atrophy of the skin and of the subcutaneous tissue and atrophy of the bone. The disease has been progressing for about a year. The prognosis is bad and the disease will probably progress, involving more extensive areas than shown in the picture. There is no history of heredity and no history in the patient of antecedent disease. She is an exceptionally healthy young servant girl who has been in this country about two years. The case will be reported more fully hereafter.

Dr. JAMES HENDRIE LLOYD exhibited

A SECTION OF THE SPINAL CORD

removed that morning from a young child in the Home for Crippled Children. The patient was one of our old

chronic cases of spinal caries of which we have quite a large number in that institution. She had large abscesses running down in front of the iliacus muscle into the groin, and another opening in the sacral region and communicating directly with the spinal canal. I removed, simply, enough of the cord to show what we have in these cases before pressure symptoms become marked.

This child, with extensive caries and marked angular curvature, had presented only the symptoms of commencing pressure on the spinal cord. She had somewhat increased knee-jerks and contractures of the legs (probably due in part to the abscesses), without distinct paralysis of either the motion or sensation, and without involvment of bladder. We find on examination of the specimen quite extensive pachymeningitis. At one point, just at the angle in the spine, where the abscess cavity opened into the canal on its anterior surface, the dura mater was perforated and partially adherent to the jagged edges of the cavity. The fact that this permitted free drainage probably explains the fact that the cord escaped as it did. The cord was bent to almost a right angle, and still preserves this shape since its removal from the body. There is slight leptomeningitis. The substance of the cord is firm. It is quite remarkable how the cord escapes for a long while in these cases; a fact which, as I have said, is probably explained in cases like the present by the establishment of drainage through a discharging sinus.

In addition to the spinal lesion, this child had tubercular deposits in the lungs and mesenteric glands; also extensive tubercular meningitis at the base of the brain. She had also amyloid kidneys.

NOTE ON THE TREATMENT OF MYXŒDEMA WITH RAW THYROID GLAND.

Dr. FRANCIS X. DERCUM.—I should like to mention that I am now treating a patient with myxœdema by feeding her with raw thyroid gland. Within ten days the patient improved so much and the hands were so shrunken that they were no longer typical. Neither is there so much swelling in the face as before. There is also improvement in the mental condition. The patient receives half a gland a day. It is chopped up with raw beef, seasoned with salt and pepper and a little onion added. It is taken without difficulty. I shall later report the results obtained.

SOME AFFECTIONS OF THE THIRD AND FIFTH CRANIAL NERVES.

I. Bilateral Oculo-Motor Paralysis.—II. Oculo-Motor Paralysis and Anæsthesia in the Distribution of the Frontal and Lachrymal Branches of the Trigeminal Nerve.—III. Trigeminal Paralysis and Spasm.—IV. Clonic Masseter Spasm or Jaw Chattering in the Aged.[1]

Cases Observed in the Department for Diseases of the Mind and Nervous System of the Philadelphia Polyclinic.

Service of Dr. CHARLES K. MILLS.

Reported by J. W. McCONNELL, M.D.,

Clinical Assistant and Instructor in Electro-Therapeutics in the Philadelphia Polyclinic; Assistant Physician to the Epileptic Hospital.

I. Bilateral Oculo-Motor Paralysis.

Case I.—W. H. O., aged 52 years; colored; married; laborer; native of Virginia; with an unknown family history; presented himself at the Clinic, April 9, 1893. He had a chancre in 1876, followed by a skin eruption. A few years later he contracted a second chancre, and subsequently had buboes and skin eruption, with falling out of the hair. He had no other illness until two years ago, when he began to suffer with pains in his legs, as though he had cramps. These pains continued for two weeks, during which time he was unable to walk. A short time after this he had a paræsthesia of the trunk which he described as feeling like a cord drawn tightly around his body. Since Christmas (1892) this sensation had been growing less in degree, although still present.

The present trouble commenced last Christmas with sharp pains starting in the frontal region, and shooting over the head to the occipital region and the back of the neck. Soon the upper lid of the right eye drooped, causing ptosis of that side, which lasted two weeks. This improved, but he noticed a similar involvement of the left eyelid, and at the same time found that he was unable to see objects in certain positions.

On examination, April 9th, he had complete ptosis of the left eye and partial ptosis of the right. Movements of the eye internally, upward, and downward, are completely lost. Both eyes are drawn into the external

canthi and are immovable in any direction. Sensation is not impaired in the slightest degree, conjunctival reflex being present in both eyes. He does not complain of pain in the head or eyes, but still has to a certain extent the girdle sensation of some time ago. Examination of the knee jerks show that the left is present and easily reinforced, and the right is absent, but reinforcement will cause its appearance to a slight degree. He has no paralysis of any other part of the body, but constantly complains of vague pains throughout his body, and an annoying numbness of the hands.

The treatment has been anti-syphilitic and electrical. A mild galvanic current has been used for over a month

with marked improvement of the right ocular muscle and some on the left. The question of a possible tabetic condition of this case is "sub-judice;" more probably he has suffered from a form of syphilitic myelo-neuritis.[2]

II.—Oculo-Motor Paralysis, and Anæsthesia in the Distribution of the Frontal and Lachrymal Branches of the Trigeminal Nerve.

Case II.—W. B. T., 60 years of age; white; male; without occupation; a native of Connecticut; came to the Clinic, March 29, 1893. Thirty years ago he contracted

a chancre, but has no positive history of further specific disease, although eight years ago he had suffered with a small sore on the right upper arm which did not tend to heal until after he commenced treatment with iodides and mercurials.

In January, 1893, he sold tickets in a small room heated by a gas stove. The cold air came through the small ticket window and was constantly striking him in the right side of the face. Within a week he was seized with sharp pain in the eyeball and right frontal region, which was followed in a short time by the appearance of an

[2] Gowers: (W. R.) Disease of the Nervous System. Am. Ed., Philadelphia, 1888, p. 614.

herpetic eruption extending from the median line over the right frontal and temporo-parietal regions, and the face immediately under the orbit, and probably involving the conjunctival surface of the eyelids. Two weeks later, the eruption continuing, sensation of burning became noticeable, commencing in the side of the nose and extending over the location of the eruption. They were abrupt and transient in character, not more than two or three in a day, but at times so severe as to cause a feeling as though the part was being scalded. At this time the patient noticed that the right upper eye-lid was paralyzed. He does not know, however, when the ptosis became complete. The right eye seemed to be somewhat sore at first, and later became very much so.

His condition March 29th was noted as follows: The remains of an herpetic eruption is still seen scattered over the right forehead and temporo-parietal regions as far back as in a line with the posterior border of the ear; it extends all over the eyelids, but does not involve the ear or cheek at all. Over this area there is complete anæsthesia; the cornea is also insensible to touch. There is complete ptosis of the right eyelid, and much inflammation of the eye, both conjunctival and tarsal. The cornea is cloudy, but there is no ulceration. The movements of the eyeball performed by the internal rectus, superior and inferior recti, and inferior oblique muscles are completely lost. Both cycloplegia and iridoplegia are present.

The chief interest in this case is the simultaneous involvement of two cranial nerves and the consequent question as to the pathological process underlying it. The complete paralysis of the oculo-motor nerve, and the incomplete loss of power of the first division of the trigeminus suggest the possibility of a double lesion. That the affection of the fifth nerve is peripheral is proved by its peculiar distribution. Ranney[3] says that "peripheral

[3] Ranney: (A. L.) Lectures on Nervous Diseases. Philadelphia 1888, p. 83.

lesions of the fifth nerve cause anæsthesia of special parts supplied by small branches or single filaments of the nerve." In this case the trouble seems to be confined solely to the frontal and lachrymal branches of the ophthalmic division of the fifth nerve.

The pathology of the condition is more difficult to determine. Erb[4] regards anæsthesia of the trigeminus as frequently of inflammatory origin. In the present instance the history of syphilitic infection, of exposure to cold, the presence of pain and trophic disturbances, and the absence of symptoms of intracranial trouble, all point to an inflammation, syphilitis or rheumatic in character, preferably the latter, affecting the nerve.

The oculo-motor nerve became affected while the trouble with the trigeminus still existed. The probability of the same local lesion causing both the oculo-motor and trigeminus paralysis has to be considered. Owing to the anatomical relations of these two nerves, the question of extension by contiguity of structure cannot be accepted, hence we look further. The history of the case shows that the patient has had syphilis, but years ago. Gowers[5] recounts a case of oculo-motor palsy, where thirty years had elapsed since the infection. He claims that the nerves may be the seat of syphilitic inflammation, or of a gumma, or may be involved in a syphilitic meningitis.

De Schweinitz[6] regards syphilis as the most frequent cause of third nerve palsy, the resulting paralysis being due to a peripheral inflammatory or gummatous change affecting the nerves at the base of the brain. Cyclo-plegia, the same authority says, is due to lesion of the trunk of the oculo-motor nerve or the anterior part of its nucleus.

Iodide of potassium was prescribed in large and increasing doses and galvanic electricity was used three

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[4] Erb: Ziemssen's Cyclopedia of the Pract. of Med. Am., Ed., Vol. XI., p. 224.

[5] Gowers: Op. cit., p. 614.

times weekly. At present, nearly two months after coming to the service, he is able to separate the lids almost fully, the external movements of the eye are nearly perfect, the internal movements are improved, but not to so great a degree as the others. The anæsthesia is better and the eruption fast disappearing.

III.—Trigeminal Paralysis and Spasm.

Case III.—J. T. W., aged 46 years; male; married; a carpenter; native of Maryland; family history good; came to the service for Nervous Diseases of the Philadelphia Polyclinic, March 22, 1893. December 1, 1892,

he was affected with an acute cold,—coryza, cough, etc. About the first week in January, 1893, he noticed for the first time that he could not chew as well as formerly. He was especially weak when trying to bite. This weakness of the muscles of mastication increased, and in the course of two or three weeks the muscles became powerless to such an extent that the lower jaw was not held in position against the upper. Ability to crack the jaws together was variable. In the latter part of February the jaw became affected with a tremor, causing chattering or clonic spasm, and occasionally the jaws would come

[6] de Schweinitz: (G. E.) Diseases of the Eye, Philadelphia, 1893, p. 528.

together involuntarily. About the beginning of March, he became unable to close the mouth except with external assistance. There was no loss of sensation.

He is a fairly well-nourished man, without any paralysis of the limbs or trunk, but with a peculiar appearance of the face. The chin is dropped, causing a separation of the teeth of about one-quarter to one-half inch, with the lower lip somewhat everted and pendulous. Attempts to bring the jaws together cause the tremor. Movements of the lips and other portions of the face supplied by the seventh nerve are preserved. Sensation is preserved. The movements of the temporal, masseter, buccinator and pterygoid muscles are all impaired.

The patient is unable to swallow food that is wholly masticated, and at the same time hold in his mouth the part that is not masticated. In order to swallow fluids he must push his mouth shut after the drink is taken, and drop his head towards his chest, else what is taken n will regurgitate through his nose. Once in a while fluids get into his trachea. Warm drinks are especially irritating. The act of swallowing is very peculiar: during it a part of the fluid seems to go down by gravity, then a spasmodic action of the muscles of the front of the neck takes place, and some goes down and some regurgitates through the nose. Apparently the movements performed by the digastric, mylo-hyoid and stylo-hyoid muscles are affected, and those of the tongue seem also to be impaired. Electrical examination of the affected muscles showed contractility to both currents without any change in the normal formula, but an unusual slowness of response, most marked when the faradic current was used.

The palatal muscles contracted to irritants somewhat imperfectly. The constrictors of the pharynx contracted perhaps a little less actively than usual; the vocal cords and arytenoids moved perfectly. In drinking he was unable to get the water far enough back for the constrictors to act. Laughing gave the upper part of his face a peculiar appearance, somewhat like the risus sardonicus.

The fields for form and color showed moderate concentric contraction.

There were no changes in the electrical reaction except as noted, which practically barred out a peripheral affection. The case seemed to be either nuclear or hysterical. The patient was put upon general building remedies with the supposition that the disease might be hysterical, or, at least, functional. Iron, quinine, and strychnine in good doses were administered internally, and were combined with local treatment, with massage and faradic electricity. After four weeks' treatment, improvement was very noticeable. He was better able to swallow liquids and the regurgitation through the nose was less marked,—in fact, often absent. Two weeks without any local treatment did not result in retrogression.

IV.—Clonic Masseter Spasm or Jaw Chattering in the Aged.

Case IV.—M. K., aged past 70 years; white; widow; native of Ireland; can tell nothing of the family history, and little of her personal history. She was well six months ago when she first noticed a "peculiar quivering sensation in the region of the stomach," which was followed by a desire to vomit, and sometimes by the complete act of vomiting. Immediately after these symptoms a movement of the lower jaw would occur, lasting less than a minute, but frequently repeated. This state of things has continued to the present time and her condition is as follows:

The woman is of worrisome disposition and can think and talk of nothing but her troubles. Her general health is poor. Her face is intermittently involved in bilateral spasm or twitching of the masseter muscles. The spasm is clonic in character and not attended with pain. The intervals of stoppage vary from a few seconds to almost a minute, and if her attention is diverted the period will be even longer. So far as is known the movements cease during sleep, but do not entirely stop when

mastication is attempted. The spasm is not very regular and an attempt to count the number of oscillations was fruitless.

This class of cases is spoken of by Gowers[7] as rare and occurring principally in old women. Romberg[8] cites a case in a woman, aged sixty-five years, in whom there was marked clonic spasm of the masseter muscles, principally the right, which always became worse towards evening and ceased during sleep.

A CONTRIBUTION TO THE STUDY OF INSANITY AND NEPHRITIS.

By AMELIA GILMORE, M.D.
Permanent Resident, Philadelphia Hospital for the Insane.

JUST now medical science is making its advance by giant strides in pathology. It is a seductive path, and no persuasion is necessary to send the flower of the medical profession into its shadiest nooks. "If nature has secrets she must give them up," they say, and to know the cause of disease is the first step toward curing it.

So etiology is deservedly at the front, and all research or observation which can throw any light on the causes of disease is welcomed. And every little helps; as some one said, "There are mites in science as well as in charity."

We are so much accustomed to following our standards, accepting without question what a recognized teacher has promulgated, that in departing from the rule we instinctively turn back to our classics, to see how our opinions have been formed, and through what paths we were led into error, if error there be.

More than a generation has passed since an authority on mental disease wrote:[1] "Insanity may originate from purely physical causes."

"Diseases of the kidneys and the anomalies with which we are as yet acquainted of the urinary secretion, do not appear to be of great importance in the etiology of mental disease. Some cases may be mentioned in which insanity probably had some connection with an affection of the kidneys, but these must be very rare, and it is impossible to give any particulars regarding them."

We agree that forty years has marked progress in the recognition of disease, its causes and effects.

As early as 1858, the best statisticians of mental disease,[2] make a carefully prepared statement that the greater proportion of insanities are due to physical

[1] Griesinger on Mental Disease, edition of 1867.
[2] Bucknill and Luke; Causes of Insanity.

rather than moral causes; but as this was a reversal of former belief, they cautiously added that "more extended statistical data are required before we can certainly conclude whether these figures are exceptional."

Further, the same authorities find that diseases of the heart, lungs, stomach may produce mental alienation, but that the kidneys are remarkably free from disease in all forms of insanity, and in their practice not one case of Bright's disease had ever been met with among the insane.

Ten years later there was little change from the earlier expressed opinions, and again another decade in which old theories held sway. Nearly every organ in the body, except the kidney was thought to act injuriously on the brain cells under some circumstances, each producing different types of insanity; and intestinal, vesical and hepatic insanity were recognized.

Mania, as a symptom of Bright's disease, was observed by Samuel Willes in Guys Hospital[3] in a number of cases, but he hesitated to assert that uraemia was the cause, on account of other pathological conditions which might have produced it, and also because other observers had not described similar effects following lesion of the kidneys.

Sankey[4] recognizes that diseases of the kidney exist in insanity, yet makes them secondary to impurities in the circulatory medium. This vitiated blood he considers is sufficient to alter the minute structure of the kidney, which in turn acts upon the heart, causing hypertrophy of the chief chamber from overaction. His vicious circle begins with the changed condition of the blood, "due to an error of assimilation."

Clonston[5] regards nephritis as an etiological factor in producing insanity, and has had several such cases under his care, and adds that they usually occur in chronic cases of Bright's disease.

More recently, Spitzka,[6] in the chapter on Somatic

[3] See Journal of Medical Science, July, 1874.
[4] Lectures on Mental Disease.
[5] Mental Diseases, 1884.
[6] Manual of Insanity, 1890.

Etiology of Insanity, enumerates diseases and injuries of the brain and its coverings, chorea, post-febrile insanity, rheumatic and phthisical insanity, valvular heart disease, the puerperal state, as causes, and says that the view has been expressed that when albumenuria coexists with the maniacal and frenzy-like explosiveness of the puerperal state, the uræmia is a collateral etiological factor and adds, that no substantial grounds exist for endorsing this view.

Bevan Lewis,[7] recognizes premature senility as a local manifestation of chronic Bright's disease, and that there is an imperfect renal function in these cases associated with chronic brain atrophy, due to extensive vascular change and fatty deterioration in the brain cells. Further than this he makes no reference to kidney disease as associated with insanity.

Now, let us consult recent and accepted writers on general medicine, to learn what is the opinion of other than alienists regarding mental symptoms following defective urinary secretion, also for the causes of nephritis.

Fagge,[8] states that albumenuria appears in certain affections of the nervous centres. There is also a brief note of delirium which may occur in uræmia. Further than this we find no reference to the involvement of the higher centres in this affection.

Strumpel,[9] "Disturbances of circulation make themselves felt in the kidneys." A general arteria-sclerosis may be the first step in producing Bright's disease. Cardiac hypertrophy the second, especially of the left ventricle. He writes that albumenuria occurs after emotional disturbances, and after epileptic attacks, or in other severe nervous conditions. In certain cases of Bright's disease, urea is retained in the blood in sufficient quantity to cause disturbance of intellection, emotional crises, incoherence, or the various symptoms of mania though the convulsions and coma of uræmia may be wanting.

Osler,[10] recognizes that there may be a sudden onset of mania in uræmia with rapidly fatal results; also that

[7] Text-book on Mental Diseases, 1890.
[8] Principles and Practices of Medicine, 1886.
[9] Pext-book of Medicine, 1887.
[10] Principles and Practices of Medicine, 1892.

delusional forms of insanity are often developed following such a pathological condition. In considering the etiology of chronic parenchymatous nephritis he mentions habitual over-eating as a frequent excitant of the disease, also degeneration of the vascular tunics, and he states that beer and alcohol are believed to lead to this form. He observes the prevalence of Bright's disease in this country, and considers that it may be accounted for by the anxieties of business and worry.

It is noteworthy that the two groups last mentioned give us precisely the conditions which are believed to give rise to a large proportion of the insanities; so that we may not assert in a given case that the nephritis was a potent factor in the production of insanity, or conversely, that the mental derangement having been induced by certain moral causes was followed by a physical disability, one organic disease causing non-resistance in other and remote organs, but we have reason to believe that in some cases the two may be produced simultaneously, though the manifestations of one may be more pronounced or more readily recognized than the other.

Again, it is so common that every physician will remember some cases where he has treated Bright's disease in its many phases and with no knowledge of tendency to insanity in the patient, he has yet come slowly and unexpectedly to recognize a positive mental decrepitude without making a logical deduction of cause and effect.

All the elements for disease lie within the human body. It is not necessary that some foreign substance or structure be introduced into it in order to disturb the harmony of its members, as is the case in all specific disease. In health chemical action changes the substances which we call food into forms of matter suitable to be taken up by the different tissues as they have need, and this power is regulated by a selective capacity inherent in each form of matter. In the constant interchange and breaking up of substance which living entails, some portion becomes useless—it is effete and no longer subserves any use in the animal economy. It must be cast off.

Again, in certain conditions other substances harmless under normal production are largely in excess of the demand and some channel is crowded with the surplus, or one organ is given an undue share of the labor of disposing of the useless material, and as a result it is exhausted while the other is disabled for lack of use, and a third suffers because of the lack of coöperation with one or both of the preceding. In the end there is disaster surely, for, though all the different parts of the organism do not suffer equally, either in kind or degree, yet the relation is so intimate—they are like children nourished under one roof, of whom one cannot feel an injury without exciting the sympathy of all.

I sometimes feel as if the adage, "All roads lead to Rome," finds its parallel in the ailments of the human body, and that all conditions of the body—all diseases, organic or specific,—may lead to insanity.

Insanity and phthisis have long been known to bear a relation.

Ovarian insanity was recognized by all the older writers and has some adherents still.

Insanity of puerperal origin is frequently observed.

Delerium of fevers is mental aberration of transient type.

Insanity of syphilitic origin is undoubted; also, that there is a true alcoholic type.

It is undisputed that the nerve lesion of general paresis is the mode of onset of that dread disease.

It was Schrœder Von der Kolk who described insanity of the colon, while disease of the liver was believed to seriously affect the mental state.

They wrote of rheumatic, choreic and gouty insanity, and still the list grew, but without including renal insanity, though two such cases were made mention of in 1871.[11]

In current literature the most notable article which has appeared is the address of Dr. Alice Bennett, State Hospital for the Insane, Norristown. Title, Insanity as

[11] Reports Royal Edinburgh Asylum.

a Symptom of Bright's Disease,[12] with a record of sixty cases. Her conclusions briefly are:

1. That affections of the kidney are very common among the insane.

2. That uræmic poisoning is one of the most frequent causes of insanity.

3. That the most prominent and constant symptom is some form of mental pain.

4. That the motor centers are specially liable to affection.

Another article was read before the New England Psychological Society, December, 1891, entitled, Kidney Disease and Insanity,[13] with conclusions:

1. Chronic nephritis is sometimes the cause of mental aberration which may be called insanity.

2. Long continued anxiety may cause albumenuria, hyalin, granular, epithelial and blood casts with accompanying œdema in some cases.

3. This kidney disease may be temporary, disappearing when the cause is removed, or the cause persisting too long may become chronic renal disease.

4. Contrary to the opinion of many observers, disease of the kidney is quite common among the insane.

In my experience insanity is more frequently associated with nephritis than with any other disease. Out of one hundred and fifty consecutive admissions to the Female Department for the Insane, Philadelphia Hospital, in 1892, 25% give evidence of kidney disease on entrance by the presence of albumen or casts, one or both in the urine. In the admissions for the present year the percentage is a little higher—27.5%[14]—and this, although the number received includes many cases of chronic insanity.

As you observe, the number is much too large to

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[12] Read before the Medical Society of the State of Pennsylvania. June 12, 1890.

[13] George T. Tuttle, M.D., McLean Asylum, Somerville, Mass., *American Journal of Insanity.*

[14] Of the same admissions, 12% had some form of cardiac compliaction, mitral lesion being the more frequent. Other physical ailments give but very small proportion.

quote severally, and a suggestive case can only be here and there cited.

In many of them the kidney was the only organ beside the brain which showed disease, though accentuated heart sounds were frequently found, while valvular lesions and atheromatous arteries were not uncommon.

In my list, all ages are represented from seventeen years for the youngest, to a case of mania at eighty years, the majority being below forty years.

The age is important, as the Bright's disease which follows a general endarteritis, occurs as a rule late in life.

As may be expected, a large proportion of these cases are quickly fatal, the complication of disease being difficult to combat, and ground is lost in the beginning by delay in bringing the person to treatment, or the insanity appears only as a late symptom of an incurable disease and the patient is brought to the hospital to die. So that of the fatal cases, one-third were under hospital care for one month or less.

The brief account of two such cases is appended:

Entry, No. 1150, P. H. Hospital record; female; aged 46 years; married.

Insanity was thought to have had a gradual onset, though it did not necessitate special treatment until some months had passed. She had labored under depression of spirits, the cause of which was unknown to her family. The melancholia took now a more definite form and she declared that she had caused the death of her son by poisoning him and she tried to poison herself.

On admission the flabby skin and muscles indicated lack of tone. The heart gave evidence of hypertrophy with accentuation of the second aortic sound. Atheroma was beginning to be felt in the radials.

Urinalysis—Sp. grav., 1024, acid—albumen in large amount; no casts observed.

There was obstinate refusal of food, and mechanical feeding was necessary. Her condition did not improve.

Uræmic coma supervened and death occurred thirty days after entrance. No autopsy.

Entry, No. 1772, P. H. record; female; single; housekeeper; age 40 years.

Previous to admission, had been for three weeks

under great mental strain, having been suddenly deprived of her home by the death of an aged relative. She lost her appetite and could not sleep. Was taken to a hospital of the city for rest treatment, and while there jumped from the second story window, causing no serious injury. Could not tell why she did it.

On admission she was in great agitation. The heart's action was strained. She could not be kept quiet, but was moving about moaning and wringing her hands constantly, presenting typical agitated melancholia.

Urinalysis—Sp. grav. 1010, acid—albumen. No casts observed.

During the second week she was actively suicidal, trying to strangle herself. There was no improvement. On the thirteenth day she died in coma. No autopsy.

But the fatal cases are only one-third of the whole. Of the remainder fully one-third recover, while the others drop into the line of the stationary cases or the chronic cases with exacerbations of mania or melancholia. Knowing the complication, a guarded prognosis is advisable, for, though the record of recoveries is good, relapses are liable to occur, a like condition producing a like result and relief may be transient.

Entry, No. 1530, P. H. Record, is a case with recovery; female; age 27 years; child's nurse; single.

Friends had noticed some slight mental change five months before she was brought to the hospital, by the exhibition of hysterical symptoms. After that she began to visit hospitals and dispensaries, receiving treatment for her stomach. But she did not improve. Slept little, and became altogether unfitted for useful living.

When admitted her physical condition was fair with accentuation of the aortics, second sound. The kidneys showed faulty secretion. Urinalysis—acid, sp. grav. 1034. No albumen or sugar, but hyaline casts.

Mentally there was well marked hypochondriasis.

The case was plain. She quickly began to mend, and convalescence was established at the end of two months. In ten weeks she was paroled, recovered. It is more than two years now with no relapse.

Entry No. 1712; female; age 38 years; widow; housekeeper.

The previous history of the case was meagre. It was said that she had been taken to St. A.'s Hospital with dysentery, and that while there the insanity had developed. There was also information that she had indulged too freely in alcoholic stimulants.

On admission she exhibited marked incoherence, with loss of memory and sense of time, while fleeting hallucinations of sight and sound were observed, and motor excitement was extreme. The first sound of the heart was booming. Respiratory murmur feeble. Urinalysis—acid, sp. grav. 1021—albumen. Epithelial casts.

After one month's treatment the mental gain was considerable. Urine examination showed no albumen or casts, sp. grav. 1012. In six weeks she was entirely reasonable, and her parole followed at the end of the second month from admission.

Nothing has been heard from her since, now eight months later.

The following case shows the tendency to relapse:

Entry No. 1764; female; age 26 years; married.

Symptoms of insanity noticed only one week before entrance by unusual conduct. Her husband being temporarily absent from home, she believed that he had forsaken her. She accused her neighbors with trying to poison her, and took the bread from her children for fear that they would be poisoned.

On admission she presented the signs of delerium grave. Took no nourishment voluntarily, was stuporous—recognized no one.

The bowel was constipated and there was urinary retention, the quantity being much diminished.

Urinalysis—sp. grav. 1030, acid. Albumen in large quantity and many broad hyaline and light granular casts.

After being two weeks under treatment, improvement was marked. She talked reasonably and took nourishment without urging. The urine showed only faint trace of albumen and no casts.

She continued to improve, showing herself gentle and affect onate. At the end of three months she became again maniacal, shouting, singing, and dancing, destructive and filthy.

At this writing, after five months' residence in hospital, she is in active mania. The urine still contains albumen in small quantity.

These cases are not exceptional. Out of the many which are suitable for illustration, they are taken almost at random.

Again, some of the cases do not exhibit nephritis as the sole physical disability, but there is found organic change in the heart or lungs, neuritis, paresis, or, specific disease, which may have its influence in producing mental aberration.

Complicated cases are not easily controlled, and the prognosis is unfavorable as regards relief from insanity. Such patients, if attacked by any intercurrent disease as influenza, dysentery, erysipelas, fevers, have little chance of recovery, and in case of epidemic disease they are very liable to be affected by it as the general systemic tone is so low.

The death register of the female department of the year 1891, shows that 14% were due to some form of nephritis, and in 1892, with a largely increased mortality, 24.1% resulted from like cause.

The records of forty-eight autopsies, for the same period, give evidence of diseased kidneys in every case save one.

Mental depression, spoken of elsewhere as belonging to these cases as a class, while it is common, does not appear in all; and, again, in some isolated cases, where the distress of mind is positive and clinical observation of the heart and local œdemas point directly to a derangement of the kidneys, the urine is searched in vain for positive evidence of the disease.

A recent writer[15] speaks of painful mental depression as the expression of "the hunger of the brain cells," without considering that the cause of the trouble may be the urea-freighted blood which cannot nourish and must destroy.

Urine of low specific gravity is very frequent among the insane, even when no albumen is present. Oxaluria is not rare and uric acid crystals are common.

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[15] Bevan Lewis (1890).

As a physical symptom, we find accentuation of the aortic second sound the usual accompaniment of parenchymatous nephritis, a common feature in all classes of the insane.

Cases of general paresis often exhibit disabled kidneys in an early stage of the disease.

Among paranoias there is no rule, but nephritis affects a large proportion. It is not prominent among puerperal cases.

Pregnant women under hospital care, having chronic interstitial nephritis, are found to come to a normal delivery as a rule, no uræmic symptoms appearing and interference is not indicated.

But besides those in whom the nephritis is demonstrated on entrance, we have still a large proportion with a like complication which is not developed until months or years of hospital residence have passed. It may be that the early symptoms were not observed, but that it is frequent among the chronic insane is positive, and the causes are patent—as have been mentioned. Frequent emotional avalanches as disturbers of circulation, which must in turn affect the chief chamber of the heart-boulmia—advancing age—general sclerosis.

I have said that it is possible that the early symptoms of this organic disease may be unobserved. All physical disease is masked in the insane, but especially is this true of nephritis, and one must be alert to catch even a single feature. Then there are pit-falls in the examination of the urinary secretion. It is a general rule in hospital work that an examination of the urine of the patient be made as soon as may be after entrance. If this analysis furnishes no pathological interest it is rarely repeated, though to judge the case by this one test is unwise. It may be only after numerous careful analyses that the existence of disease can be shown. But the labor attending these examinations is considerable. The time spent over a few specimens daily can be reckoned by hours, and the number of the patients is so large it becomes a physical impossibility for the hospital physician to make as frequent examinations as would be desirable.

Supposing the early analysis of the urine has been made with normal result, yet if the positive demonstration of diseased renal tissue is made after a few weeks or months of residence in the hospital, it would seem to be conclusive evidence that the condition had been the same or nearly so, at the earlier date, but the proof was wanting.

Careless methods of work give poor results. Examinations that promised much may yield nothing in the hands of one who takes little interest in the subject.

A recent medical assistant in the insane wards, getting his first practical knowledge of insanity, said to me: "You have many cases of nephritis; how is it that I find nothing about it in the books on mental disease." This may be explained as the influence of the former teachers which is to-day still felt by the older alienists, who are now in a position to direct the work of others. It is not they who come in direct contact with the patient. They are slow to see new facts brought out by chemical and microscopical tests which they had neglected. They were instructed that the kidney was an unimportant organ in the etiology of insanity, instead of serving as chief health officer over the whole body—and that "all forms of injurious matter, which circulate in the blood, must be eliminated from the body in great part of the kidneys." (Strümpell).

In conclusion, we assert that it is not unwarranted to search for physical causes of mental aberration, but that it is clearly indicated by the premises. The mind is the superstructure of the body. That insanity should be produced by the disease of an organ which is known to carry off substances injurious to the body if retained seems reasonable. If the kidney fail in its office the blood becomes charged with urea. Not all brains possess a like sensitiveness to adverse influences, or to changes in pabulum. Not all stomachs are dyspeptic.

A belief that there is some physical cause for mental alienation is supported by reason and precedent. Our logic demands for the effect a cause, and though we deal with the highest type of matter—the trained cerebral cell

—it is certain that it will follow the inevitable law when its function is interfered with. Like sweet bells out of tune, the harmony is turned to discord. A variation in the normal elements of sustenance may be sufficient for this. Urea retained, the watery elements of the blood increased —the nerve tension lost, and disaster follows, the foundation being insecure, something less than an earthquake may shatter the tower.

Organic change was observed in the brain of a person dying in uræmic coma.[16]

There was found to be a destructive process going on in the nerve cells of the cortex, and especially in the nerve fibers nearest the white matter. The latter had entirely disappeared in some places, while phagocytic action was plain, the debris taking the shape of black granules with which the leucocytes were loaded.

That insanity should be a symptom, even though no destruction of tissue is found, needs no comment.

Again, whether tissue change, which may accompany mental derangement is seen as well in the walls of minute vessels as in the brain cells and may manifest itself in the changed secretion of the urine is a point yet to be determined; but it is consistent, with present theory, to say that the nephritis may directly follow the insanity—the excessive emotional storm giving extra work to the arterial system with the consequent hypertrophy of the great chamber of the heart.

That nephritis is associated with insanity in a large number of cases cannot be doubted, and the fact deserves serious consideration.

The corollary follows:

1. That insanity and nephritis may be concomitant, both resulting from the same causal condition.

2. That nephritis should be expected as a tolerably certain sequence to the perturbed forms of insanity.

3. That among the nervous symptoms of Bright's disease, must be reckoned mania and melancholia.

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[16] The examination was made by Dr. A. B. Macallum, University of Toronto, vide article in *Canadian Practitioner*, April, 1889, "What is Uræmia?"

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